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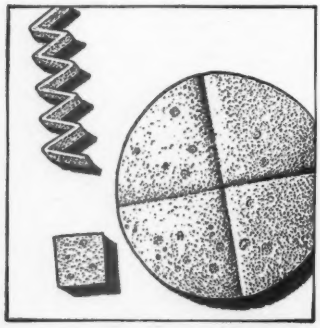

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ADULT INTUSSUSCEPTION AND TUBERCULOUS ENTERITIS

REPORT OF A CASE*

GEORGE C. RASCH, M.D.

Indianapolis, Ind.

Intussusception is usually a disease of infancy and childhood; only a relatively few cases occurring in adult life. The condition in the adult differs from that in infants since the causative factor in infancy is rarely found, while often a causative factor may be identified in adult cases. In reviewing cases of intussusception from the James Whitcomb Riley Hospital of the Indiana University Medical Center, Moore[11] found a causative factor in only 3 of 37 patients 2 years of age or younger and in 5 of 6 children ranging in age from 4 to 18 years. Tumors, Meckel's diverticula, dysentery, typhoid ulcer and occasionally tuberculous enteritis have been found to be factors in adult intussusception. Although the occurrence of tuberculosis of the small bowel with intussusception has been noted, only a few such cases have been reported. For this reason the following case is described.

The patient, a white man 44 years old, complaining of severe abdominal pain of five days duration was admitted to the Indianapolis Veterans Administration Hospital in November 1952. The pain had started suddenly and increased rapidly in severity. Nausea and vomiting had been present for three days and no flatus had been passed for two days. There had been present rather vague abdominal pain for several months prior to the acute illness. In addition, the patient had lost 25 pounds in the past year and had noted marked fatigue for many months. There was no history of previous surgery or gastrointestinal symptoms. Examination showed the patient to be acutely ill with a temperature of 99.6 F. and

* From the Department of Surgery, Indianapolis Veterans Administration Hospital and Indiana University Medical Center. Reviewed by the Dean's Committee and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are a result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

pulse of 100 per minute. Other findings included marked abdominal distension with high pitched bowel sounds. The lower abdomen was tender to percussion and palpation, particularly on the right side. No masses were palpable and no abdominal rigidity was present. Dark blood was found on the examining finger after rectal examination. The patient was moderately dehydrated. The white blood count was 11,300 leukocytes per cu. mm. with 83 per cent neutrophils. Urinalysis was negative. A roentgenogram of the abdomen showed what was thought to be a typical picture of low small bowel obstruction. A roentgenogram of the chest revealed infiltrative densities bilaterally, probably due to active pulmonary tuberculosis.

A diagnosis of complete small bowel obstruction—probably strangulated—was made. Adhesive tuberculous peritonitis and intussusception were considered with other conditions in the differential diagnosis. An operation was done on the day of admission. The abdomen was opened through a right lower quadrant transrectus incision. An irreducible ileocecal intussusception was found and resection of the intussusception was done followed by an end to end ileo-ascending colostomy.

The patient made an uneventful recovery. Sputum examinations were made and many acid-fast bacilli were found. Prior to transfer to another facility for care of the pulmonary tuberculosis, para-aminosalicylic acid and streptomycin were started. At the time of transfer the patient was eating well and had normal bowel movements.

Pathologic examination of the resected specimen showed an intussusceptum 4.5 cm. long, indurated and edematous. Microscopic examination of the wall of the intussusceptum showed, in addition to gangrenous changes, caseation and tubercle formation. No acid fast bacilli were found. However, with the history of active pulmonary tuberculosis and the presence of caseation and tubercles in the wall of the intussusceptum, a diagnosis of tuberculosis of intussuscepted ileum was made.

DISCUSSION

Intussusception has been generally recognized as a disease primarily of infancy and childhood and cases in adults are rather uncommon. Edington[4] stated that 68 per cent of cases of intussusception occurred in the first year of life. According to Nichols,[12] 75 per cent of such cases occur in infants under 2 years of age. Iason[8] noted that only 5 per cent of all cases were in adults. In a series of 151 cases reviewed by Kahle,[9] 18.9 per cent of cases of intussusception were found in patients over 16 years of age, the oldest patient being 63 years of age.

Martin and Dennis[10] described four basic types of intussusception: ileocolic, ileocecal, colocolic, and ileo-ileal or enteric. Donhauser and Kelly[3] found that the ileo-ileal variety was the commonest in adults.

The clinical course of intussusception in adults varies from that usually seen in infants. Observers agree that in adult cases symptoms may be vague and gradual in onset compared to the acute onset and course usual in the infant. According to Brown and Michels,[2] the adult case often resembles chronic recur-

rent small bowel obstruction. Abdominal cramping is the most frequent symptom. A mass is not usually palpable and blood in the stool seems to be infrequent. Cases with acute symptoms may, of course, occur in adults. However, adults may stand delay of surgical treatment longer than infants. Kahle[9] found that in a group of adults with intussusception operated upon before 48 hours no patient died, but in infants when operation was deferred for over 24 hours, all of the patients died. Moore[11] found no deaths in a group of infants and children operated upon within 24 hours, while the mortality rate rose appreciably in a group treated at a later period.

It is a common observation that a causative factor, such as a Meckel's diverticulum or a tumor, may be found in adult cases of intussusception. Tuberculosis as a factor has been reported only occasionally. Eliot and Corscaden,[5] in a review in 1911, of 300 cases of adult intussusception, mention 5 cases of tuberculous enteritis associated with intussusception. Bolker and Hamilton[1] and Gunter and Trout[6] reported single cases very similar to mine. Neither of their patients survived resection.

Although the examination for acid fast bacilli in the ileal wall was negative, I believe that the diagnosis of tuberculous enteritis in my case is correct. The histologic picture of the ileal lesion is typical of a tuberculous inflammation. According to Hoon and co-workers,[7] ileocecal tuberculosis may be identified by the presence of caseation and tubercles. Acid fast organisms may be found in only 30 per cent of specimens examined. The occurrence of this typical intestinal lesion in association with active pulmonary tuberculosis would serve further to establish the diagnosis since ileocecal tuberculosis occurs most frequently in conjunction with pulmonary tuberculosis.[7]

The treatment of intussusception in adults, as in infants, should be reduction if possible. Definitive surgical treatment for the causative factor will, of course, have to be done far more often in adults than in infants. Resection of involved bowel in an irreducible intussusception may be necessary, as in my case. Although the mortality rate in reported cases of resection for intussusception associated with tuberculous enteritis has been high, the outcome in this case would suggest that recent advances in preoperative and postoperative care of such seriously ill patients may make such necessary resections a relatively safe procedure. This should be true for resection for irreducible adult intussusception, regardless of cause, provided operation is not unduly delayed.

SUMMARY

A case of irreducible intussusception associated with tuberculous enteritis occurring in a white man is reported. The intussusception was treated successfully by resection. Adult intussusception is discussed briefly.

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CARDIAC ARREST

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INTRODUCTION

Cardiac arrest is cessation of circulation as judged by the usual methods of physical examination. It implies an element of suddenness as well as of unexpectedness. This distinguishes it from that group of conditions wherein the heart is gradually failing. Cardiac arrest seems to be increasing in frequency. This may be due to the fact that there are now larger numbers of major intrathoracic and abdominal operations and that poorer risk patients are now submitted to such surgical interventions. Many of these people, only a decade or so ago, would have been considered unfit for operation; many are affected by one of the conditions often considered to predispose to cardiac standstill. The data in our own institution seem to indicate that the occurrence of this condition is approximately 0.1 per cent of all operations.

The occurrence of cardiac arrest is not limited to major thoracic or upper abdominal operations. It may and does occur in otorhinologic, ophthalmologic, orthopedic and other procedures. It therefore is mandatory that every room in which any kind of operation is done be prepared for this emergency. Preparedness hinges upon the organization of the operating room and all of its personnel —physicians, nurses and attendants—and that they be educated in the regimen for this particular emergency. Such organization includes the training of personnel in the diagnosis and therapy and the instant availability of the equipment necessary for the management of cardiac arrest. The importance of *immediate diagnosis* and *immediate action* can not be overemphasized. Careful and continuous observation by the surgical team and especially by the anesthesiologist, during all operative procedures, is of the utmost importance. Careless observation of the patient prior to the onset of cardiac arrest and thus a delay in starting adequate circulation will markedly diminish the chances of return to normal function. It has been stated that complete hypoxia of approximately three minutes is the maximum time for resuscitation without permanent damage to the brain.[1, 7] Almost any normal heart that goes into cardiac arrest can be rescued provided there has not been too long a delay in diagnosis and treatment. This is particularly true if the arrest has occurred in *standstill* rather than in ventricu-

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lar fibrillation, and if there has not been a chronic state of hypoxia preceding the arrest. The greater this preceding hypoxia or the longer its duration, the shorter will be the period of total oxygen lack compatible with recovery.

Once arrest has occurred, it is too late to organize for therapy. Instruments designated for this purpose should have been put aside in all places of medical therapy where this condition may conceivably occur. Apart from the main operating rooms, such places as the tonsil and genito-urinary rooms, the bronchoscopic and x-ray rooms should also have the equipment at hand. This equipment, together with the outline of therapy, should be discussed and practiced with the entire operating room staff. The minimum equipment available should be a scalpel, a retractor or retractors, surgical hemostats, sutures, rib shears, needles, syringes, sponges, scissors, and apparatus for giving artificial respiration with oxygen. The necessary drugs include 1 per cent procaine, 1:1000 epinephrine, calcium chloride 10 per cent, vasopressors, and oxygen. This routine must not only be discussed, but it should also be typed out and readily available in every location where cardiac arrest may occur.

ETIOLOGY

I. Background factors
   1. Degenerative heart disease
   2. Vascular heart disease

II. Precipitating factors
   1. *Hypoxia and/or hypercarria*
      a. Shock
      b. Obstruction to respiration
      c. Depressed and ineffective respiration and circulation
   2. *Hypercarbia*
      Soda lime exhaustion
   3. *Reflexes*
      a. Failure to block
      b. Light anesthesia
      c. Trauma to viscera
   4. *Acute myocardial failure*
      a. Shock
      b. Coronary accidents
      c. Deep anesthesia
   5. *Mechanical factors*
      a. Inflow and outflow obstruction
         Acute
         Gradual

Background Factors

Beck has pointed out that the probability of cardiac arrest is greater in the patient with heart disease than in those who have a normal heart.[2] The normal heart is capable of withstanding many insults which in the diseased heart may

well precipitate an arrest. It is therefore important that we know the cardiac status of all patients prior to the time of surgery. One should obtain an adequate history and determine the physical status, and know the exact nature of any possible heart disease. Factors such as heart strain, toxicity, increased myocardial irritability, metabolic disease, all add to the probability of arrest. Such conditions as coronary disease, myocarditis or degenerative myocarditis, valvular heart disease, especially with regurgitation, heart block, cardiac dilatation, myocardial hypertrophy and atrophy are other factors which predispose.[6] Marked tachycardia and bradycardia are also considered predisposing factors by some. Any disease which prevents adequate cardiac filling, such as the mediastinal syndrome, stab wounds of the heart, pericardial effusion, and constrictive pericarditis, come into the same category. Diseases of the respiratory system which lead to poor exchange and which diminish oxygenation are likewise factors. Diseases which interfere with respiration, such as large accumulations of fluid, gas or a tumor in the abdomen and adiposity, will also increase the probability of arrest. All these diseases are background factors. Their presence will markedly enhance the chances of arrest but their absence on the other hand is no guarantee of immunity against such a catastrophy. Against these background mechanisms, we must now consider a number of precipitating factors. They are primarily hypoxia, hypercarbia, reflexes, acute myocardial failure, and mechanical causes.

Precipitating Factors

Hypoxia. Respiratory depression or respiratory obstruction from whatever cause will tend to make the heart more irritable because of hypoxia. Excess depth of anesthesia or excess narcotics, each depressing respiration, or paralysis of respiratory muscles due to overcurarization may cause such hypoxia, if respiration is not supplemented. In these instances, it is not the agents which are to blame but rather the failure of those administering them to maintain adequate ventilation. Poor exchange from obstruction or from secretions in the respiratory tree are other factors of great importance in lowering tissue oxygen tension. Shock, hypotension, hypovolemia or anemia may also be responsible for this diminished oxygen tension. The absence of cyanosis is of very little importance except that when cyanosis is present it is definite evidence of interference with the cardiovascular mechanism. Many positions in common use during operations markedly impair cardiorespiratory function and may thus be important factors in the causation of cardiac arrest. Among the most injurious are the steep Trendelenburg, the acute lithotomy, the lateral and prone jackknife positions. In children depression of respiratory efficiency is even more easily produced than in adults, especially when the weight of the drapes is placed directly on the small child. When the adult-type anesthetic equipment is used for the small child, it not only causes a respiratory inefficiency but hypercarbia as well. Noisy respiration is indicative of obstruction and should be interpreted as a sign of impending disaster.

Hypercarbia. Hypercarbia or an increase in carbon dioxide is observed in al-

most all of the conditions enumerated under hypoxia. Recent research has pointed out that carbon dioxide in marked excess is present much more frequently than had hitherto been suspected.[4] Studies have shown that concentrations of carbon dioxide are often almost unbelievably high. In the *open chest* case for example, unless one takes special precautions, hypercarbia is almost always present.[4] Factors of importance are not only those mentioned before but also additional ones such as poor or exhausted soda lime, mechanical defects in the anesthesia machine, and the use of the open drop mask. It has been shown that carbon dioxide can build up to dangerous concentrations with this technic, which has so often been considered the essence of safety as well as of simplicity.

Reflexes. Stimulation such as manipulation of a fracture, tugging on an abdominal viscus, and insertion of an endotracheal tube, are relatively noninjurious in those people who have a normal cardiorespiratory mechanism. But in those who have hypoxia, hypercarbia, or both, the possibility of damage from these stimuli is markedly increased. This is especially true if there is present some concomitant disease which has led to an increasedly irritable, degenerative, or depressed myocardium. Hypoxia alone is certainly not deisrable and a reflex in the presence of a mild degree of hypoxia is dangerous; yet, the superimposition of carbon dioxide excess markedly increases the chance of cardiac arrest. In patients with cardiac disease this danger becomes even more acute, and it becomes exceedingly important to forestall as much reflex activity as possible. To this end the surgeon should adequately block with a local anesthetic all afferent stimuli from the field in which he is working.

Adequate general anesthesia is of great importance in the prevention of cardiac arrest. A light plane of anesthesia can be truly hazardous, and depth must always be adequate for the work at hand at any particular moment. This becomes exceedingly important when one is working in richly innervated areas such as the upper abdomen and the hilus of the lung. The correct plane is always the lightest one consistent with the work at hand for a *definite patient* at a *definite time.* It is never static and must be changed depending upon the patient's reaction to stimulation.

Acute Myocardial Failure. Acute myocardial failure may occur with the use of cocaine, chloroform, ethyl chloride or similar myotoxic drugs. Mismanaged spinal anesthesia or unduly deep anesthesia in a patient with a diminished blood volume may cause acute myocardial failure. It may occur in patients in whom cyclopropane is being administered and when drugs such as epinephrine or posterior pituitary extract are added. It may occur simply as a result of excessively deep anesthesia. This factor is probably of much more importance than is commonly suspected. Acute myocardial failure may be due to shock, or to extensive hemorrhage or because adequate cardiac filling is interfered with due to the release of large masses from the abdomen with consequent diminished cardiac return; or myocardial failure may follow marked increase of peripheral resistance with intense vasoconstriction due to excess doses of vasopressors.

Mechanical Factors. Tension, traction, and torsion applied extrinsically or intrinsically to the heart or great vessels may mechanically interfere with the

inflow or outflow of blood, and thus may lead to cardiac arrest. Such mechanical interference with heart action may be acute, as in embolism of large vessels or of the heart itself, and in mechanical compression or kinking due to improper placement of retractors and packs, or due to sudden hemorrhage into the pericardial sac. Other mechanical factors may be of a more gradual nature, as in tamponade of heart from blood or serous effusion in the pericardium, or as in constrictive pericarditis. Mediastinal neoplasms may produce inflow or outflow obstruction either acutely or in a more gradual manner.

PREVENTION OF CARDIAC ARREST

The prevention of cardiac arrest is by far more important than its treatment. It begins with an adequate history and physical examination. Thus knowledge may be gained of possible existing factors which may predispose the patient to cardiac arrest; these conditions should be treated as far as this is possible. The patient should be brought to the operating room in an optimal state; hemoglobin and blood volume should be adequate. Unfortunately the blood volume is often unknown and one may be misled by relatively adequate hemoglobin; in reality this has only minor significance, the total blood volume being far more important than hemoglobin determinations.

Cardiac efficiency should be brought to an optimum by the use of cardiac glucosides, quinidine, procaine amide, and diuretics as indicated. The patient should be in adequate hydration at the time of operation. Any conditions contributing to the previously discussed etiologic factors should be corrected. Thus, adequate respiratory exchange with adequate oxygen tension are prerequisites of all anesthesia. There is no excuse for any technic which is based upon low oxygen tension. The importance of carbon dioxide as an etiologic factor of cardiac arrest cannot be overemphasized.

Gentleness in handling tissues is of great importance. An effective block with a local anesthetic agent in the sensitive areas is important also. Not only does this prevent reflexes but it enables maintenance of anesthesia in a much lighter plane and the patient benefits from this balanced type of light anesthesia. Such blocks imply an adequate knowledge of the part and of the afferent pathways. Atropinization also is important in the prevention of reflexes. Atropine is accepted generally as an agent which tends to prevent vagal type reflexes and as such may help in preventing cardiac arrest. Unfortunately, frequently—especially in children—the dose of atropine is more symbolic than real.

Blood pressure per se is not necessarily indicative of the state of the vascular system; respiration and pulse frequently are much more vital signs. Air hunger, rapid or labored respiration, and chin tug are very definite and important signs of difficulty. These signs of hypoxia and hypercarbia must be avoided if we are to reduce the incidence of cardiac arrest.

Teamwork is as essential in the prevention of cardiac arrest as it is during resuscitation. *The surgeon should at all times be kept informed of the condition of his patient.* Cyanosis, shock, poor exchange, and poor blood color, must always be relayed to him and the surgeon in turn should keep the anesthetist informed

of his observations. It is the failure to work as a team that so frequently leads to disaster.

Diagnosis

Absence of blood pressure does not necessarily signify cardiac arrest. An imperceptible peripheral pulse, although not absolutely diagnostic of arrest, is sufficiently significant as to warrant such a tentative diagnosis until proved otherwise. A very rapid check is made for evidence of pulsation in other peripheral areas, and if no such evidence is forthcoming, the surgeon is informed of the gravity of the situation. If the chest or abdomen are open he will be able to confirm or refute the diagnosis by palpating the large vessels; or in thoracic cases, by inspecting the heart itself. If this is not possible, a therapeutic test is made by injecting 2 mg. of neosynephrine with atropine sulphate 0.8 mg. (gr. 1/75) in 20 cc. solution into one of the large vessels. Cardiovascular collapse is far more common than cardiac arrest, and the injection of these two substances will frequently be sufficient to improve circulation, at least, to a point where evidence of it reappears. It also may aid in breaking a cardiovascular inhibition of vagal origin and thus help in restoring circulation. All of these investigations should not exceed 45 seconds, and if evidence of circulation is still lacking, steps are taken to enter the thoracic cavity. However, it is well to observe the skin incision in the thorax for the presence of active arterial bleeding; if such is encountered, it signifies that the heart is not in arrest and thoracotomy is deferred. If no bleeding is encountered, or if dark venous and capillary blood issue forth only passively, the thoracic cavity is entered without delay.

Treatment

Treatment consists of two distinctively different phases. The first phase which allows no delay aims at the restoration of the oxygen transport system by means of artificial respiration and manual systole. This should be accomplished within three minutes of arrest, or at least within two minutes of making the diagnosis. In other words, little over one minute is allowed to elapse between suspicion of arrest and incision into the chest. The second phase is one of re-establishment of spontaneous cardiac activity. As oxygenation of the vital organs is being maintained by artificial means, during this period, this phase of resuscitation is relatively less urgent.

Phase I: The instant cardiac arrest is even suspected, a number of actions are instituted forthwith.

1. One person takes charge and remains in charge of treatment throughout the period of resuscitation. It matters little whether this person is the surgeon or the anesthesiologist, as long as he is well acquainted with the routine and with the various means of treatment. Continuous changing from one person to another in the control of the patient will certainly be harmful.

2. Anesthetic agents are discontinued and oxygen is administered in a flow of 10 to 20 liters per minute in order to wash out anesthetic agents and carbon dioxide. If not already in position, an endotracheal tube must be passed as soon

as conditions permit; but in the early stages, provided the airway is free and the respiratory exchange adequate, it may be best to wait for a more opportune time for the passage of the endotracheal tube. No degree of respiratory obstruction should be tolerated, neither must passage of the endotracheal tube interfere with artificial respiration. In the absence of an endotracheal tube, oxygen may find its way into the stomach, leading to extreme gastric distention and further cardiac embarrassment. Therefore, after tracheal intubation a Levine or similar tube should be passed into the stomach to empty it of air and oxygen, but this too must not be allowed to interfere with artificial respiration. Unless gastric dilatation is extreme, this step may well be deferred. The patient's respirations are assisted, or artificial respiration is given if spontaneous breathing has ceased. Undue pressure on the breathing bag is avoided, because overdistention of the lungs diminishes cardiac filling; inspiratory pressure on the bag is of short duration; the resting phase is long. A heart will not start unless one is adequately oxygenating the lungs which in turn supplies the necessary oxygen to the myocardium.

3. Some person present in the operating room and not required for other duties, begins to call out the time every 15 seconds, and reports every minute how much time has passed. This count too begins the very instant when evidence of circulation can no longer be found.

4. Any factors that may have contributed to the arrest are immediately discontinued. Such factors, usually of a reflex nature, include manipulations of the hilus of the lung, tugs on the mesentery, spreading of the ribs, and so forth.

5. The operating table may be tilted into a 5 to 7 degree head-down position, in order to facilitate arterial blood flow to the brain and venous return to the heart. Inclines steeper than this lead to cardiac embarrassment and do more harm than good.

6. It may be wise for the surgeon—if the abdomen is open—to pass his hand over the left lobe of the liver and feel for heart action. On occasions the mere mechanical stimulation of the ventricle may be enough to re-start the heart. Cases have been reported where a vigorous thump on the chest has started a heart. However, one should look upon this with a degree of suspicion. Yet, in a very small child the mechanical stimulus from marked compression of the chest may re-initiate cardiac action.

The diagnosis of cardiac arrest having been definitely made, no time is lost to artificially restore the oxygen transport system to the vital organs, in particular to the brain. This can be accomplished effectively only by entering the thoracic cavity. If the abdomen is open, mechanical stimulation from below may temporarily be made, while the chest is being opened, but it is not to take the place of artificial circulation by the thoracic route. The abdominal approach is less efficient; also it is impossible to differentiate with certainty between cardiac *standstill* and ventricular fibrillation through this approach. The thoracic approach is the only one which allows the performance of adequate manual artificial circulation.

Time is not wasted in securing sterile drapes or in surgical preparation of the

skin. An antiseptic may be applied to the skin, if this causes no undue delay. The incision is made anterolaterally over the fifth or sixth left rib, lateral to the internal mammary vessels. This site will avoid trauma to the internal mammary vessels and will minimize the danger of injury to the heart itself. The incision then may be extended medially and costal cartilages are cut. If no active bleeding is encountered with the skin incision, the chest is opened; the hand is introduced; and the diagnosis of *standstill* or of ventricular fibrillation is made. Manual compression of the heart immediately is begun. Frequently a few such compressions will re-start the cardiac cycle.

In carrying out manual systole, the heart is grasped in the hand and is compressed in such a way as to empty both chambers. The manoeuvre is carried out from the wrist and consists of a milking, flat-handed compression which tends to shift blood from the apex of the heart to its base. In essence, the criterion of efficient manual systole is the palpation of an adequate peripheral pulse with each compression. Blood, properly oxygenated in the artificially ventilated lung, is thus moved to the vital organs of the body; this brings to a conclusion the first phase of cardiac resuscitation.

Unless the steps just described, and the meaning of each, are thoroughly understood, there appears to be a tendency on the part of many physicians to do a number of other things as well. Unfortunately most of these do no good under the circumstances; they waste valuable time and some of them may actually do harm. To avoid a few of the more common errors:

DON'T wait for an electrocardiogram.
DON'T needle the heart.
DON'T give analeptics.
DON'T compress the chest, nor dilate the rectum.
DON'T give intra-arterial transfusions.
DON'T give epinephrine during this phase of resuscitation.
DON'T give digitalis. None of the cardiac glucosides act fast enough to be of value during the time available.

Phase II: This phase of resuscitation, while maintaining the oxygen transport system by artificial means, is concerned with the re-establishment of cardiac automaticity. It is doubtful whether any one person can efficiently carry on manual systole for longer than two or three minutes. Hence a second person should be available to take his turn. A rate of approximately 70 per minute is probably optimal, but must ultimately depend upon adequate cardiac filling during relaxation.

The conduct of the second phase will depend upon whether the heart is in ventricular fibrillation, or whether *standstill* has occurred. The state of fibrillation must first be converted into *standstill* for two reasons. Fibrillation exhausts the meager oxygen supply to the myocardium and thus increases the hypoxia of the heart muscle, making it less and less responsive and diminishing the chances of successful resuscitation. Effective manual systole with adequate oxygenation of vital organs, including the heart itself, is markedly impaired in the presence of fibrillation. Hence defibrillation is a matter of urgency; delay may render the myocardium nonresponsive to stimulation of any kind.

A number of ways and means are available to defibrillate a heart, and they should be tried in succession until one is successful. Procaine—because it diminishes myocardial irritability—is first administered. Our preference is for a quick injection of 20 cc. of 1 per cent procaine into one of the large arm veins; or injection may be made into one of the large intrathoracic veins, provided this does not interfere with manual systole. If fibrillation persists, the injection may be repeated a number of times after an interval of one to two minutes. It is far better to have a heart that is overly depressed by large quantities of procaine than to have a heart which is in fibrillation, and which becomes increasingly hypoxic and unresponsive to later attempts at resuscitation. If procaine therapy is of no avail, recourse should be had to one of the electrical defibrillators. There are a number of such apparatus on the market; they should deliver a surge of 110 volts, 60 cycles, and approximately 1½ amperes, but not less than $^{8}/_{10}$ ampere. The electrodes—approximately 7 cm. in diameter, with or without suction cups—are placed, one anteriorly and one posteriorly, flat against the heart.[3] It is important that they be firmly in contact with as large an area of the heart as is possible. This increases the benefit of stimulation and reduces the hazard of thermal injury to the heart muscle. *Under no circumstances must the electrodes touch each other as this would short circuit the machine.* The shortest shock possible is applied. Some of the apparatus on the market have a shock duration of $^{1}/_{10}$ to $^{5}/_{10}$ of a second; some even deliver shocks of one second duration; others are provided with manual control by which any length of stimulation can be applied. With some of the defibrillators pulsations can be applied rhythmically at a rate of 80 per minute or so, and it has been suggested that the instrument may be used in place of manual systole.[3] In our hands this method has not been as effective as manual systole as judged by the quality of the pulse, color of skin and myocardium, and physiologic signs of oxygenation such as size of the pupils.

In the arrested heart which has not been in fibrillation and which has not been allowed to become unduly hypoxic, manual systole alone may suffice to restore spontaneous cardiac activity. The heart in arrest is a depressed heart and procaine alone is therefore as contraindicated as is epinephrine in the hyperirritable, fibrillating heart. Neither should epinephrine be used in a heart with relatively good tone or when one believes that the heart is just about ready to take over by itself. This is indicated by occasional or by frequent beats independent of manual systole. If epinephrine is decided upon, a solution (½ cc. epinephrine 1:1000 in 20 cc. water) may be injected into one of the large veins or into the cavity of the left auricle after the heart has been protected by procaine. Of this, 200 mg. is given intravenously and 5 to 10 cc. of a 1 per cent solution is injected into the pericardial sac.

If the heart fails to respond to this therapy, an attempt may be made to restart it by using the electrical defibrillator. Calcium chloride 2 to 10 cc. of a 10 per cent solution injected into the left ventricle is frequently of value in initiating cardiac rhythm,[5] or in reinforcing the beat if it appears to be weak. In the defibrillated heart epinephrine is extremely hazardous; therefore electrical stimulation and calcium should be tried first; only if this line of therapy is of no avail, should epinephrine be carefully tried.

While these attempts at re-starting the heart are made, manual systole is persevered with. It may be interrupted only for the very brief moments while the defibrillator electrodes are being applied. It is not necessary to open the pericardium, unless manual systole is continued for a prolonged period, or direct inspection of the heart muscle is necessary in order to eliminate definitely the presence of fibrillation. In very rare instances a final decision cannot be arrived at without the electrocardiogram after manual systole has been started. By and large, however, the heart arrested in systole is in standstill, while the large, flabby heart is or has been in fibrillation. In order to assist the supply of blood to the brain, compression of the descending aorta may be found to be of much help. It constantly must be remembered that oxygenation of heart and brain are the primary concern of the moment.

During the period of resuscitation, intravenous fluids are kept running at a slow rate to provide an avenue for intravenous medication. Great care must be taken to avoid circulatory overload, also not to administer solutions which may increase cerebral edema. If there has been extensive hemorrhage, however, blood replacement is carried out during this time. Serum albumen and plasma expanders may be helpful in those instances in which adequate blood is not available.

After the heart has been started, a period of at least 10 minutes is allowed to elapse during which time the heart can be observed before the chest is closed. After 10 minutes of efficient circulation, the heart is not likely to stop again. If a peripheral pulse cannot be obtained despite spontaneous cardiac activity, rapidly acting glucosides, such as ouabein or strophantin K are indicated. Vasopressors are administered with extreme caution because the increased blood pressure increases the work load of the heart and again may arrest it.

The patient is still further observed in the operating room and is not moved until one is quite certain that movement will not be harmful.

After-Care

The after-care of these patients may be considered under three headings: factors concerning adequate respiratory exchange, those of circulatory function, and supportive measures.

1. Adequate respiratory exchange implies a good tidal volume and administration of oxygen. Artificial respiration, if necessary, may be given by the use of mechanical devices. When spontaneous respiration is adequate, high concentrations of well humidified oxygen are administered, preferably by a mask. No depressants or narcotics of any kind should be used. Restlessness is more likely to be due to a degree of cerebral hypoxia than to pain; therefore narcotics would surely be harmful. No analeptics should be used. The secretions from the tracheobronchial tree must be quickly and carefully removed whenever they accumulate. Tracheostomy should be done if there is obstruction above the larnyx or if secretions are excessive. It is preferable that the endotracheal tube be left in place for at least a matter of 24 to 36 hours.

2. Cardiac glucosides may be given to improve circulatory function. The blood pressure should be maintained at an adequate level by the very cautious use of

vasopressors such as norepinephrine, neosynephrine, and methoxamine in minimal quantities. The blood pressure should not be raised any higher than is absolutely needed. Physiologic sodium chloride solution should not be administered because of the fear of edema of the brain. One may use a slight head-up position to encourage diminution of edema in the brain but this should be done only when the circulation to the brain is adequate. The stellate ganglion block is of nebulous value; it may be used if one so desires. Adequate fluids and electrolytes must be administered. The use of albumin, dextran or other hypertonic substances to aid in the dehydration of the brain is of some value. Blood should be given when indicated; diuretics may be valuable in diminishing the cerebral edema.

3. Supportive measures include adequate nutrition by tube or intravenously, if necessary. Carbohydrates are very important for the nutrition of the brain. High vitamin B and C may be administered intravenously. Antibiotics should be given as indicated. Aspiration of vomitus must be avoided at all costs, and chest complications must be assiduously guarded against. A competent team should be in charge of the minute-to-minute care of the patient. Routine nursing care must be of the highest caliber and includes attention to bladder and bowel, the care of the eyes, and the avoidance of bed sores.

CONCLUSIONS

In conclusion, it may be stated that cardiac arrest is an ever present hazard in the operating room. It is more apt to occur when operations affect those areas of the body which have greater degree of reflex activity, particularly in persons with predisposing diseases. Hypoxia, hypercarbia, and reflexes may precipitate the arrest. Early recognition and the early treatment will enable us to save many of these patients, but it requires careful preparation and organization and careful attention to the details of resuscitation once it has occurred. This will enable resuscitation of a higher percentage of patients.

The entire problem should be discussed at meetings. Equipment and organization must be planned beforehand. Study commissions should be appointed by various localities to encourage research, development, and statistics which would enable us to gather more information as to the causation of this condition. Also it would aid in the development for better instrumentarium for early detection of the predisposing factors as well as the development of newer and better equipment for its treatment.

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CANCER OF THE BREAST

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The etiology of cancer of the breast is still unknown, but according to H. L. Stewart,[5] the frequency of cancer of the breast varies with different circumstances of demography. He states that based upon the deaths per 100,000 female population of the United States, cancer of the breast and of the uterus account for about an equal number of deaths (uterus 24.15 and breast 24.65). In Denmark the morbidity figure for cancer of the breast is lower in the rural areas and higher in the cities. In the United States, uterine cancer is 60 per cent more frequent in Negroes than in whites, whereas cancer of the breast and ovary are more frequent in whites.

Japanese women, compared to United States women, showed the same number of deaths from uterine cancer, but only one-sixth as many deaths from breast cancer. In India, the Hindu women who are poor, marry early, and have many children; have 50 per cent of all cancer arising in the cervix; while in the upper classes, women who have married later and have had fewer children, have 50 per cent of the primary cancers in the breast.

Similarly, in England, breast cancer is said to be more common in women living under better economic conditions. Stewart states that single women have a higher rate of cancer of the breast than cancer of the cervix, while in married women the reverse is true. In France it has been reported that less cancer of the breast is found in women with a large number of children than is found in women with a smaller number of children. However, childless married women after the age of 40 seem to have more breast cancer than the women who were never married.

From these statistics it can be judged that there are a variety of different conditions which affect the incidence of cancer. Among these are: marriage, childbirth, hormones, heredity, and social status.

In neoplasms of the breast, the endocrine factor still demands the chief consideration in spite of the recent work on the milk factor and the virus influence. The majority of the benign lesions such as adenofibromas, intracanalicular adenoses, intraductal papillomas, and cystic disease, are all proliferative hyperplasias of the mammary tissue and are all probably produced by hormonal influences. Statistics have shown that there is a higher incidence of cancer of the breast with these pre-existing lesions than in patients without such pathology.

Nathanson[3] states that the effect of hormones on breast cancer seriously challenges the theory of autonomy of cancer cells. Observations have shown that neoplastic cells are susceptible to direct attack through change in their

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environment by agents which are essentially physiologic in action. Careful and continued observations of the effect of hormones on susceptible tumors should lead to an increased understanding of the mechanism of growth processes.

The diagnosis of cancer of the breast should not be difficult and should always be made early if a few simple rules are followed. The earliest symptoms are: a lump or small mass in the breast, or an abnormal discharge from the nipple. Unfortunately, the patient frequently delays coming to the doctor until the mass has increased considerably in size and possibly has metastasized. Education of the public is very rapid, and breast lesions are now being seen by the doctor earlier than ever before. Robbins and associates,[4] in reporting experiences at Memorial Hospital in New York, found that in cancer of the breast there was almost 50 per cent reduction between 1946 and 1948 in the delay in time from the first symptom and the first visit to the doctor. This is most encouraging. It is the duty of the physician who first sees the patient to start her on the road to proper treatment.

Abnormal discharge from the nipple is one of the most urgent signs for consultation. In practically all of these cases the discharge is due to some neoplasm. Donnelly,[1] in reviewing 219 such cases of nipple discharges found that 45 per cent were caused by malignant lesions, 30 per cent by intraductal papillomas, 22 per cent by chronic cystic mastitis, and only 3 per cent by other causes such as retention cysts, galactoceles and inflammation. He also noted that while sanguineous discharge was most frequently seen to come from malignant conditions, malignancy was also found in many cases that had serous or yellow drainage. From personal experience and reports such as Donnelly's it is apparent that any discharge from a nonlactating breast, whether a mass is palpable or not, deserves immediate attention and no temporizing treatment should be countenanced.

Before undertaking any therapy of a breast lesion, the diagnosis must be confirmed by microscopic examination. In my opinion and in the opinion of Stout[6] and many others, an open biopsy section is the one of choice, as it is believed that aspiration biopsy specimen and examination of nipple secretions is inaccurate. It is most essential to have a quick frozen section done while the patient is under anesthesia, and if a malignancy is present, to do a radical amputation immediately. In certain doubtful cases, it may be advisable to close the wound and do the definitive treatment the next day if malignancy is found on examination of the permanent microscopic section. However, this will be infrequent and the delay should never be more than one or two days.

If, at biopsy, malignancy is found, the wound should be completely closed and the radical operation started after a complete new preparation of the skin and change of drapes, gowns, gloves, and instruments. In making the incision for biopsy section, it is best to make it directly over the tumor, and if the tumor is small and discrete, the entire tumor should be removed with a small margin of normal breast tissue. If the tumor is benign, no further incision is necessary and the operation is complete after closure of the wound. The incision for biopsy section should never be used as a part of the incision of the radical amputation.

This incision should be excised with the skin, breast, and underlying structures. Ideally, the incision for biopsy section should be in the center of the skin removed at the radical operation.

There is almost complete unanimity of opinion that a radical amputation of the breast, in those cases which do not have remote metastases, is the treatment of choice. Obviously, in a patient with metastases to the bones or the lungs, such an extensive operation would be contraindicated. In cases that are apparently malignant before operation, in addition to the routine history, laboratory studies and physical examination, a roentgenographic examination should be made of the ribs and lung fields to rule out the possibility of metastases in those regions. In all cases, except those with severe systemic diseases and distant metastases, a properly executed radical amputation of the breast seems to give the best end-results, particularly when combined with roentgen ray and hormone therapy. In recent years there have been only one or two authors who claim that in their clinics results equal to those obtained by radical amputation have been obtained by simple amputation followed by intensive roentgen ray therapy.

I shall not attempt to describe the technic of radical amputation in this paper. Suffice it to say that it should include removal of all the breast tissue and its overlying skin, with the removal of the pectoralis major and minor muscles by division close to their insertions, and careful dissection of the axilla with its nodes and contents, leaving only the larger vessels and nerves. However, if these latter structures are involved, one should not hesitate to include them in the dissection. The dissection is carried medially to the midline so that the fascia over the sternum and a small margin of the fascia of the opposite pectoral muscles is included in the dissection. It then extends downward to include the upper portion of the fascia of the two recti muscles, laterally to the latissimus dorsi muscle; to include part of its fascia and the fascia of the serratus anterior muscle. In our opinion, the majority of these cases can be done by careful undercutting of the skin flaps, leaving a minimal amount of subcutaneous tissue attached to the flaps so that the wound can be closed under moderate tension, but without skin grafts. It has been found advisable to make small stab wounds in the skin flaps which give more relaxation and also prevent venous congestion. A rubber tissue drain is placed in the lower angle of the wound and a catheter is placed in the axilla and brought out through a stab wound in the outer flap. The wound is first dressed with one strip of vaseline gauze and over this is applied an abundant firm dressing. Suction is placed on the catheter at intervals which produces a negative pressure and collapse of the dead space in the axilla. Since using this method we have found that there has been less postoperative drainage and the wounds have healed more readily. It has been our impression that in keeping down the collection of serum in the axilla by this catheter suction, there have been fewer cases of postoperative edema of the arm. This method of drainage was first proposed by David Murphey[2] of Tampa, Florida.

In those patients who have microscopic evidence of metastasis in the axilla, we have followed the practice of giving postoperative roentgenotherapy over

the areas of metastases, but we have not given postoperative roentgenotherapy in those cases in which the malignancy was confined to the breast.

Wangensteen[8] thinks that the currently practiced radical operation for cancer of the breast is essentially an incomplete operation for patients exhibiting involvement of the axillary lymph nodes since approximately 60 per cent of such patients who have axillary involvement will show regional nodes beyond the reach of the conventional radical operation. He advocates a super-radical operation which consists of the conventional radical mastectomy, and in addition, removal of the supraclavicular lymph nodes, the parasternal lymph node chain with a dissection of the lymph bearing tissue of the anterior mediastinum. He states that this is an extensive operation and cannot be broken into stages. To date, insufficient numbers of cases have been done and the lapse of time has been too short to evaluate this procedure.

Urban[7] also believes that the nodes along the internal mammary chain should be removed with the primary operation. He states that it is well known that the primary lymph node deposits are in the axillary and internal mammary lymph node chains and it is this second deposit that produces so many recurrences. He quotes R. S. Handley that 60 per cent of the inner quadrant mammary lesions will have metastases to the internal mammary nodes, while 20 per cent of the outer quadrant lesions will show this spread.

Urban advocates a procedure which consists essentially of resection of full thickness of the chest wall surrounding the internal mammary lymph node chain from the first rib to the fifth interspace and from the costochondral junction of the ribs to the mid-third of the sternum. All of this tissue is removed in continuity with the radical mastectomy. The bony wall defect is repaired with fascia lata grafts sutured in place under moderate tension in order to minimize paradoxical motion of the chest wall.

White,[9] in commenting on cancer of the breast in pregnancy, recognizes this serious combination, and because of his excellent 10 year survival rate in recent cases, feels justified in continuing radical treatment in these cases. However, he has found that carcinoma of the breast during lactation has been fatal in all of his cases. In commenting on the recent extensive radical operation, he states that Sauerbruck and others attempted this years ago and gave it up, but called attention to the work of Sampson Handley, Jr., which has shown involvement of the internal mammary chain in numerous cases, but thinks that to date not enough material has been gathered to draw any definite conclusions.

It is known that estrogen has a stimulating effect on both normal and malignant breast tissue, and it has been shown that large doses of estrogen will produce breast cancer in experimental animals.

Since 1937 it has been our practice to remove surgically the ovaries of patients with carcinoma of the breast who are still in the premenopausal stage. At first, we did surgical castration of some of these patients and roentgen ray castration of others. It is our experience, however, that surgical castration is more certain and can be easily done at the time of operation on the breast. Then, too, some of our patients who were castrated by roentgen ray menstruated a year or more

later. It is believed, therefore, that if the ovaries are to be rendered completely functionless, it is better to remove them surgically than to destroy their function by radiation.

The results have been so satisfactory with castration at the time of primary operation that it has been continued and has been extended to those cases that are one or two years postmenopausal. Surgical removal of the ovaries in a patient who has not had previous abdominal operations nor extensive pelvic pathology is an easy and simple procedure. If it is to be done at the time of the mastectomy, while the assistant is closing the breast wound, the operator makes a short midline incision through which the ovaries can be delivered; their ligaments clamped, the ovaries removed, and the pedicles sutured. This procedure usually can be finished before the breast operation has been completed.

Since 1937 we have operated upon 303 successive cases of breast cancer in women, and of this number, 66 have had surgical removal of their ovaries at the time of radical amputation of the breast. While our series of 66 cases is small, in the Stage I carcinoma of the breast we have had 94 per cent five-year survivals and in those with metastases outside of the breast we have had 63 per cent five-year survivals. There has been no complication or contraindication to this operation, and we plan to continue the practice. One of the chief criticisms has been early menopausal symptoms or decrease in libido. The menopausal symptoms have been controlled without difficulty by sedatives or the use of androgens, and there has been no appreciable change in libido.

If, for some reason such as the general condition of the patient or extensive pelvic pathology, bilateral oophorectomy would be a complicated or extensive operation, I believe that castration by roentgen ray would be the procedure of choice. Complete suppression or neutralization of the estrogenic substance by androgens has some advocates, but seems impractical to us because of the uncertainty of the required dose and the results. If androgens were used, they would have to be continued until well past the expected menopause.

Androgens reduce the pain and also the size of the metastatic lesions in patients with recurrent carcinoma of the breast. This is particularly true when the metastases are in the bony structures. Surgical removal of the ovaries in such cases, if the patient is still in the premenopausal period, will reduce the estrogenic secretion and will have essentially the same effect as the administration of androgens and will render the administration of androgens more effective; as less will be required to neutralize the effect of the ovarian secretions.

Wangensteen, Urban and others, have shown the high percentage of cases that are clinically Stage I are actually Stage II or higher. It is estimated that there is an error of at least 30 per cent in the clinical classification of the breast cancer. This error has been determined by dissection and microscopic study of the surgical specimen. The error must be larger, for undoubtedly there are many minute collections of cells which are missed in the pathologic study of these specimens.

Local recurrence is usually estimated at 10 per cent, more or less, which means that 90 per cent of the recurrences are due to cells that have already metasta-

sized to areas beyond the operative field at the time of operation. To lower the overall percentage of recurrences we must either remove these other metastases by some type of super-radical operative procedure or destroy these cells by roentgen ray.

It is known that estrogens stimulate normal breast tissue and malignant cells of the breast in premenopausal patients. If malignant breast tissue cells, which have been left in the body inadvertently, are suddenly deprived of their estrogenic stimulus, there is every reason to believe that they will atrophy or lie dormant for longer periods than if this stimulus were continued. This atrophy is well known to cases with large or obvious metastases.

More time must elapse before we can draw any final conclusions as to the value of these newer procedures. Just as hormone therapy and castration in the male has prolonged the survival rate in patients with carcinoma of the prostate, castration of the female with carcinoma of the breast has prolonged the survival rate in our small series of patients. This is an adjunct procedure and deserves further study and consideration.

SUMMARY

Cancer of the breast is influenced by many different factors such as marriage, childbirth, hormones, and social status.

The best results are being obtained by radical mastectomy in the applicable cases, but the percentage of recurrence is still too high.

In premenopausal patients, who develop cancer of the breast, surgical castration at the time of the radical operation improves and prolongs the survival rate.

The radical amputation with postoperative radiation, in cases known to have axillary or parasternal metastases, seems to give promise of better results than the most extensive super-radical mastectomy.

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SURGICAL TREATMENT OF SKIN CANCER

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Skin cancer is the most common malignancy in the Southwest.[3, 8] Although it is usually considered no serious threat to life, if it is not recognized and adequately treated, the consequences are as certain as those of a rapidly growing cancer involving vital organs. The number of patients with, and the deaths from hopelessly advanced skin cancer in a community, directly reflects the standards of medical care practiced in that community.[9]

Exposure to sunlight is considered the most potent etiologic factor in skin cancer. That exposed areas receive the most actinic rays accounts for the most common sites of skin cancer, notably, the face, the ears, the neck and the dorsal surfaces of the hands. Forty per cent of the patients treated in the Missouri Cancer Hospital[1] have malignancies of the skin. The climate and rural population of Oklahoma, which is similar to that of Missouri, constitutes an analogous chronicity of exposure and incidence of skin cancer. However, in spite of the high incidence, skin cancer does not present a problem of the magnitude of cancers elsewhere in the body. Their readily accessible sites and slow growing tendencies should, and usually do, obviate any cause for procrastination in diagnosis and treatment beyond the curative stage.

As in cancer of all types, complete eradication of the tumor and prevention of recurrence is the primary objective and the FIRST concern. This may be accomplished by the use of escharotics, electrocoagulation, sharp excision or radiotherapy, the choice depending upon the professional skill of the available therapist as well as the most adaptable method of eradication. Irradiation and surgery are now considered the two principal methods for combating skin cancer. Prior to the debut of cancer surgery, advanced or radioresistant cancers signaled ultimate and early death of the patient. With more aggressive surgical management, many of these patients are now being salvaged.

It is exceedingly difficult to formulate any standard basis for the selection of surgery over radiotherapy in the treatment of any specific type of skin cancer. Available experts in either or both fields vary in each community. Ideally, in determining priority of treatment, the opinions of both the radiotherapist and the surgeon is to the best interest of the patient.

Eradication of uncomplicated skin cancers about the head and neck by surgical excision may result in distortion of facial features and skin grafts of obviously poor color match. In such instances, surgeons must admit that irradiation definitely offers superior advantages. When surgical excision and irradiation are considered equally effective in the destruction of the cancer and professional skill of the therapists is equally competent, preference should be given to the method which produces the best cosmetic result.

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There are many situations, however, where irradiation is ineffective and the problem is primarily one of saving life. Therefore, surgery is superior in such instances. Purposely omitting cancer of the lip, this discussion concerns the advantage of surgery over equally competently administered radiotherapy in the treatment of squamous cell or basal cell carcinoma of the skin.

In degrees of superiority, the advantages of surgery over radiotherapy in the treatment of skin cancer are:

I. *Absolute or definite* in those cases in which saving life is the principal concern and the cosmetic result is a secondary matter.

II. *Relative* in those cases with cancers on unexposed areas, with thin skin, or overlying radiosensitive structures.

III. *Contingent or incidental* advantages in those cases where the indications are poorly defined.

I. ABSOLUTE OR DEFINITE ADVANTAGES OF SURGICAL ERADICATION

1. Radioresistant Lesions

When a skin cancer has proved its ability to survive a normally cancerocidal dose of radiotherapy, repeated courses can only intensify the problem and, therefore, are to be condemned. Without surgical excision—completely removing the cancer field—a residual smoldering basal cell cancer about the face will eventually and insidiously extend to vital structures. Pierce et al[7] advise surgical excision of basal cell carcinomas about the nose and orbit when not completely eradicated by one course of irradiation therapy. In the treatment of all malignancies—if the first therapy is ineffective—the ultimate prognosis is made worse. The incidence of metastasis following unsuccessful treatment of squamous cell carcinoma of the hand is invariably increased.[5] There is every indication that this is true in other skin cancers.[4]

2. Cancers Invading Supporting Structures

When skin cancer—usually the rodent ulcer—has invaded bone or cartilage by direct extension, the only means of complete eradication lies in radical excision, frequently to the extent of destroying facial features.[2] Exenteration of the orbit, destruction of the nose, removal of the palate or sacrifice of other contiguous structures, even to the point of mutilation in certain instances, is a matter of assurance rather than assumption that nonapparent deep smoldering activity will not eventually thwart all previous efforts at treatment and render a hopeless prognosis. Hospital files throughout the country over the past years are replete with records of patients who have received repeated courses of radiotherapy while this type of cancer was stealthily advancing into bone or cartilage, many to an inoperable stage and to death. Basal cell cancer about the nose and orbit has a predilection for rigid supporting structures.

3. Carcinomatous Extension into Orifices

When a cancerous process has extended into orifices, especially the orbit and perhaps to a lesser extent around the external auditory canal, the depth of orbital

FIG. 1. *A*. Basal cell carcinoma arising in the medial canthus of the left eye in a 37-year-old woman. Several courses of radiotherapy over an eight year period were ineffective. *B*. Wide cautery excision and exenteration of the orbit was necessitated by deep extent of the tumor. *C*. Plastic repair of defect accomplished two years later by pedicle flap. This case demonstrates all of the first three absolute indications for surgical treatment.

involvement and invasion of the retro-orbital bone—frequently unsuspected—is impossible to determine by preliminary examination (fig. 1). Hence, adequate depth of irradiation is problematical, and only by surgical eradication of the tumor and potential areas of extension can the hazardous threat of further carcinomatous invasion to surrounding solid structures be eliminated.

4. Cancer Arising in Radiation Therapy Burns

When carcinoma has developed in an old radiation therapy burn, surgical excision with the added problem of grafting over the area of scarring and decreased blood supply is far more successful than any attempt to eradicate the lesion by other methods. Attention to meticulous details in grafting, and particularly the use of split grafts with on-lay pressure dressings, usually proves successful and produces the best results.

5. Cancer Arising in Thermal Burns

When cancer has developed in chronic ulceration of an old thermal burn, the scarred and fibrotic overlying and bordering skin is unsuitable for irradiation. The extent of avascularity is such that only wide and deep surgical excision will insure a vascular bed adequate for successful skin grafting. Split thickness grafts offer the best means of permanent repair.

6. Metastatic Cancer

Despite many reports to the contrary, surgical removal en bloc is the most suitable method for complete abolition of lymph node metastases from skin or any other type of cancer. Ackerman[1] has reported cervical gland metastases in 5 per cent of skin cancers of the head and neck; axillary metastases in approximately 15 per cent of skin cancers of the upper extremities and, a higher percentage of inguinal metastases from skin cancer of the lower extremities.

7. Malignant Melanomas

The treacherous metastatic tendencies and the radioresistant nature of malignant melanoma is an absolute indication for wide excision of the primary lesion. If the area of lymphatic drainage is predictable, en bloc lymph node dissection yields additional protection against regional recurrence. Likewise, to facilitate adequate eradication when justifiable, preliminary excision and microscopic examination of all suspicious pigmented lesions—as stressed by Lockhart[6]—is the only means of detecting that relatively high incidence of obscure melanomas in pigmented lesions of the hands, the feet and the genitalia and a lesser incidence in pigmented lesions elsewhere on the body surface.

II. RELATIVELY SUPERIOR ADVANTAGES OF SURGICAL ERADICATION

1. When regional lymph node metastases are in close proximity with the primary melanoma or skin cancer, surgical removal in continuity causes little additional scarring and offers the advantage of simplicity and finality by en bloc eradication of the primary tumor, the regional nodes and, particularly, the intervening lymphatics.

2. That the location of skin lesions predicate the type of therapy is confirmed by the fact that cancers on unexposed skin surfaces are less radiosensitive or radiocurable than such lesions on exposed surfaces, particularly those around the face. Furthermore, the tendency to necrosis following cancerocidal doses of roentgen or radium therapy to lesions on unexposed skin surfaces contradicts

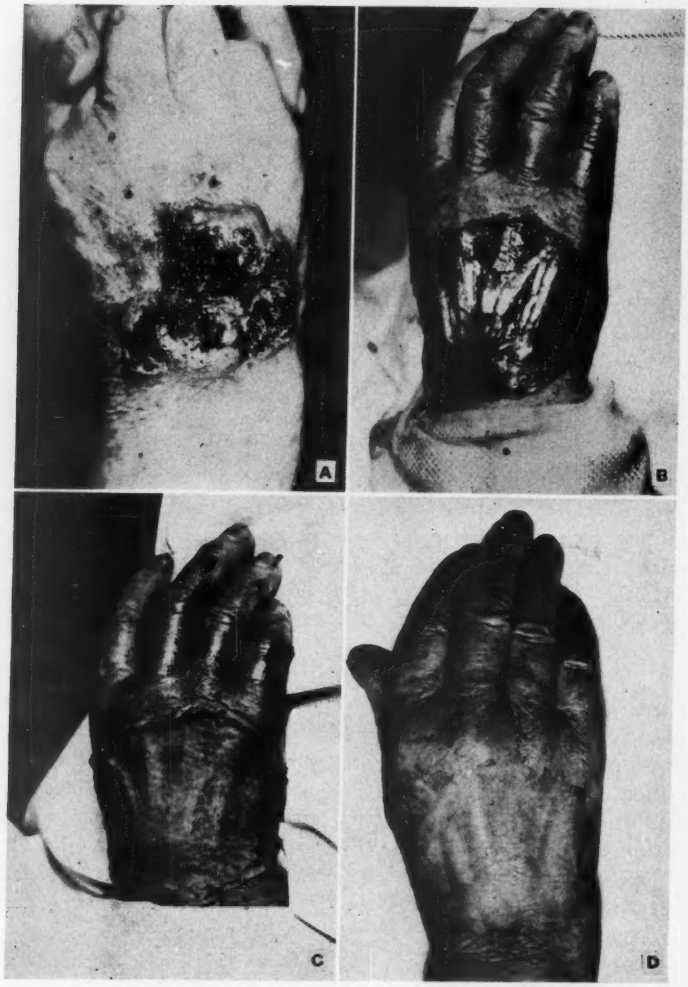

FIG. 2. *A, B, C, D.* Squamous cell carcinoma of the dorsum of hand in a 71-year-old woman. Treated by wide excision and split thickness skin graft with excellent cosmetic and functional result.

their use. Last, the objection to surgical scarring is outweighed by the *unexposed* location.

3. The skin over different areas of the body varies in its tolerance to roentgen ray. Likewise, skin cancers in different areas of the body vary in their sensitivity to roentgen rays. When supporting or subcutaneous tissue is scant and vascularity of the tumor bed is obviously poor (fig. 2), radionecrosis and chronic ulceration are prone to develop. On the dorsum of the hand, the scalp, the ear, and to a

lesser extent, the alar cartilage of the nose, there is a very small margin of differentiation between the dose of irradiation for cancerocidal effect and the amount leading to irradiation burn and subsequent necrosis. Although the occasional superficial exophytic growth may be successfully treated by irradiation, surgical excision of the more common ulcerating type of lesions in these areas is a means of avoiding such complications as radiation necrosis, chronic ulceration, tendon fixation and limitation of motion of the hand, radiation chondritis of the ear (fig. 3), or painful scarring.

4. When cancer is located in areas where the skin is exceptionally thin, for example the foreskin of the penis or the skin covering the vulva, cancerocidal doses of radiation therapy may be followed by marked atrophy of the skin, telangiectasia, pain and, frequently, recurrence of the carcinoma. Surgical excision of such lesions circumvents these complications.

FIG. 3. Squamous cell carcinoma of ear. Type of segmental resection done for cancer with fixation to cartilage. The defect resulting is minimal, producing a reduction in size of the ear.

5. Surgical excision is a practical and absolute method of eradicating precancerous lesions in toto. It is particularly incontestable in the treatment of leukoplakia of the vulva, or in the treatment of Bowen's Disease, which many consider to be carcinoma in situ. Also, when skin carcinoma is surrounded by large areas of keratosis, wide excision of the entire area and grafting is protection against radiation necrosis of the skin from overlapping of fields by repeated courses of radiation therapy.

III. CONTINGENT OR INCIDENTAL ADVANTAGES OF SURGICAL EXCISION

In certain instances, surgical excision and irradiation may be considered equally effective, although, contingent or incidental factors favoring surgical excision are:

1. When rapidity of healing is a major concern.
2. When a small lesion is situated in one of the natural folds of the face, the scar from excision is usually invisible.
3. When rapid abolition of the primary lesion may prevent subsequent metastasis.
4. When accompanying lymphadenopathy is present, more accurate and earlier differentiation between lymphadenitis and metastasis is facilitated.

5. When the cancerous lesion is large and the extent unpredictable, microscopic examination of the border and depth of the excised specimen influences further therapy.
6. When skin lesions are irradiated without microscopic examination, the presence of malignancy is always doubtful and the sequelae from irradiation continue.
7. When loss of time and limited economic resources are of major concern to the patient.
8. When repeated visits for irradiation therapy are prohibitive to the patient.

DISCUSSION

Re-emphasizing, when surgery and irradiation are otherwise considered equally effective in eradicating skin cancer, preference is denoted by the method promising the best cosmetic result.

Irradiation unquestionably offers the best cosmetic effect in the treatment of uncomplicated cancer about the face.

Surgery finds its principle use in the treatment of recurrent, residual, or complicated skin cancers and cancer on unexposed areas of the skin, irrespective of the cosmetic effect.

Group I. *Absolute or definite indications for surgery*, the primary problem is one of saving life. Complicated cancer demands the most aggressive means of attack. Surgery, limited only by structures vital to life, takes precedence regardless of any cosmetic effect.

Group II. *Relatively Superior Indications* is directed chiefly to cancer on unexposed skin with its lack of resistance to cancerocidal radiation therapy.

Group III. *Qualitative Superiority* refers to those instances where surgery offers only slight or questionable advantage over irradiation.

This discussion is not an attempt to depreciate the value of irradiation in the treatment of skin cancer. However, it seeks to define specific instances in which surgical treatment should be given precedence over irradiation.

CONCLUSIONS

The advantages of surgical excision of skin cancer may be divided into three groups of decreasing importance as listed:

I. *Absolute indications or definite advantages:*
   1. Radioresistent lesions.
   2. Cancers invading supporting structures.
   3. Extension into orifices.
   4. Cancers developing in radiation burns.
   5. Cancers arising in thermal burns.
   6. Metastatic cancer.
   7. Malignant melanomas.

II. *Relative superior advantages:*
   1. Cancers in locations permitting en bloc excision of primary lesions and metastases.

2. Cancers on unexposed areas.
3. Cancers over areas with poor *tumor bed.*
4. Cancers developing within thin skin.
5. Precancerous lesions.

III. *Contingent or incidental advantages of surgical excision:*

1. Rapidity of healing.
2. Small cancers of face may leave negligible scars.
3. Rapid abolition of primary tumor.
4. Removal of primary may aid differentiation of lymphadenopathy.
5. Sampling of borders in large lesions may influence further therapy.
6. Material available on all skin lesions for microscopic study.
7. Loss of time minimal.
8. Visits reduced to minimum.

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FEMORAL SHAFT FRACTURES TREATED WITH KUNTSCHER'S INTRAMEDULLARY NAILS AND SUPPLEMENTARY PARHAM-MARTIN BANDS*

A PRELIMINARY REPORT

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Although intramedullary nailing is accepted as the treatment of choice for transverse and slightly oblique fractures of the femoral shaft that are located 10 cm. from the tip of the greater trochanter and 10 to 12 cm. from the knee joint line, it is not generally used for sharply oblique, spiral oblique, butterfly, and variously comminuted fractures of the shaft of the femur.

Intramedullary fixation of fractures of the femoral shaft, as of all bones, is an elective procedure for which the indications, technic, advantages and limitations are ably discussed in Böhler's Book[1] and Street's articles,[5, 6] and in the American Academy of Orthopedic Surgery's Fracture and Traumatic Surgery Committee report.[4]

Böhler states that some spiral fractures, with a long butterfly fragment, are unsuited for intramedullary nailing. Such fractures are exemplified by our cases 5 and 9. Other long oblique femoral shaft fractures are treated by Böhler with intramedullary nails and adjunctive wire loops. Cases 3 and 6 in our group are similar to 2 cases demonstrated in one of his published series. In another of his examples, the primary intramedullary nail fixation of a long oblique fracture through the distal one-third of the femoral shaft was supplemented by means of a wire loop placed about the fracture. The wire loop subsequently separated. A satisfactory result was obtained through the medium of a plaster spica. Böhler believes that in this instance, a second wire loop would have prevented the breaking of the first.

When the indication exists, Böhler utilizes loops of wire for the binding of tibial grafts to femurs in which fractures are primarily immobilized by means of intramedullary nails. Street has instituted the use of supplementary screws for the fastening together of fragments around intramedullary nails in femurs, when additional support is required.[5]

Recently, we have adopted the use of the intramedullary nail with supplemental Parham-Martin bands, as the treatment of choice for sharply oblique, spiral oblique, butterfly and various comminuted fractures of the femoral shaft.

Material and Methods. Nine fractures of the femoral shaft, other than transverse and slightly oblique ones, have been treated in our series with intramedul-

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lary nails and adjunctive Parham-Martin bands. The results are reported herein. Of these fractures, 2, in the immediate subtrochanteric region, are sharply oblique; 2, near the midshaft, are butterfly fractures; 2 are long spiral fractures, 1 of the upper and 1 of the lower shaft; 2 are variously comminuted fractures; and 1 is a previously plated butterfly fracture with marked angulation.

Küntscher's SMO (low carbon molybdenum) stainless steel cloverleaf intramedullary nails and SMO (low carbon molybdenum) stainless steel Parham-Martin bands were used in all cases. Hansen-Street nails will be used for future cases.

Henry's anterolateral muscle-separating incision was used in all cases. Exact hemostasis, insofar as possible, is believed to be an important factor in the prevention of wound complications.

The patients are kept in balanced elevation (equivalent to balanced traction without actual traction) following operation, until the postoperative reaction has subsided and the soft tissue healing is well under way. The time interval varied from two to four weeks, depending upon the individual patient. The first patient treated by this method was kept in bed for six weeks, but it is believed that nothing was gained thereby. Whether or not the patient is allowed partial weight-bearing from the beginning of ambulation depends upon the impression of the observer as to the stability of the fracture after operation, and/or upon the dependability of the patient in following instructions.

Results. All operative wounds healed per primam without complications. Five patients began full weight-bearing in three to nine months following operation; 2 were operated upon too recently for full weight-bearing; 1 patient died; and 1 cannot be followed. The final evaluation with reference to range of knee flexion cannot be made, but flexion does continue to improve in each case that can be followed.

The fractures of 3 patients (one not illustrated) are completely healed; those of the remaining 5, either because of location of the fracture or continued callus production and organization, are expected to heal.

There have been two complications. One patient died in irreversible shock, immediately following a preoperative enema on the eve of the proposed second nailing. This was the only bilateral femoral shaft fracture. Both grossly and microscopically, autopsy failed to show an anatomical cause of death. The brain, lungs and kidneys were studied extensively for fat emboli, but none was found. The other complication was the failure to extract the nail from the femur of the patient of case no. 6, after seven months. The extracting eye was split after 2 cm. of extraction. The nail was cut off at the greater trochanter for comfort's sake. In the future, the nails will be permitted to remain in place for a year or longer, other factors being equal.

CASE REPORTS

Case 1. (Fig. 1.) T. T. was a 27-year-old Negro man.
Fracture: Sharply oblique, subtrochanteric, right femur with refracture, after five months of skeletal traction.
Date of Operation: Jan. 8, 1951.

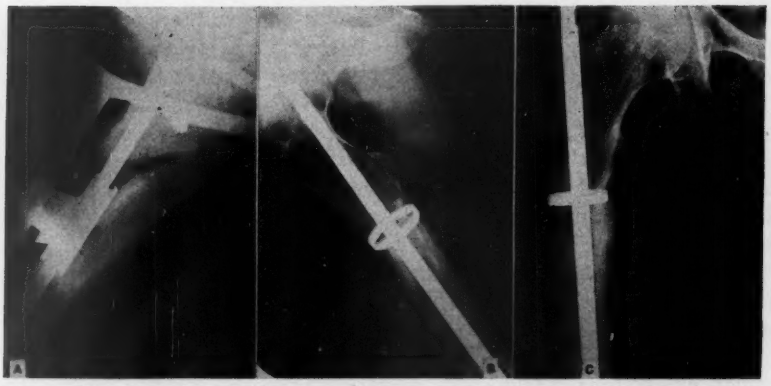

FIG. 1. Case 1. (a) A sharply oblique subtrochanteric fracture after five months of traction, with moderate callus formation just prior to refracture. (b and c) The same fracture with complete obliteration of the fracture line, 12½ months postoperative just prior to removal of the nail.

FIG. 2. Case 2. (a) A recent sharply oblique comminuted subtrochanteric fracture. (b) The same fracture immediately postoperative.

Weight-bearing: Nonweight-bearing with crutches, six weeks postoperative. Partial weight-bearing with crutches, two months postoperative. Full weight-bearing, nine months postoperative.
Knee Motion: From 80 to 180 degrees, 12½ months postoperative.
Remarks: Patient had considerable trouble with renal calculi until recently. Fracture completely healed and nail removed.

Case 2. (Fig. 2.) H. T. was a 31-year-old white man.
Fracture: Sharply oblique, comminuted, subtrochanteric fracture of the right femur.
Date of Operation: May 6, 1952.
Weight-bearing: Crutches with nonweight-bearing, one month postoperative.
Knee Motion: From 70 to 180 degrees, one month postoperative.
Remarks: Multiple attached fragments should provide good callus potential.

FIG. 3. Case 3. (a) A recent spiral oblique fracture of the upper femoral shaft. (b and c) The same fracture immediately postoperative.

FIG. 4. Case 4. (a) A recent comminuted fracture of the mid-femoral shaft. (b and c) The same fracture seven months postoperative. Minor fragments completely fused. Organization of exuberant callus incomplete. Fracture line not obliterated.

Case 3. (Fig. 3.) G. W. was a 59-year-old Negro man.
Fracture: Spiral-oblique, comminuted, subtrochanteric fracture of the right femur.
Date of Operation: May 21, 1952.
Weight-bearing: Partial weight-bearing with crutches, one month postoperative.
Knee Motion: From 90 to 180 degrees, at two months postoperative.
Remarks: Patient has failed to respond to requests to appear for follow-up examination.

Case 4. (Fig. 4.) M. S. was a 30-year-old Negro man.
Fracture: Compound, comminuted fracture of the lower right mid-femur.
Date of Operation: Aug. 21, 1951.
Weight-bearing: Nonweight-bearing with crutches, one month postoperative. Partial weight-bearing with crutches, four months postoperative. Unrestricted weight-bearing, seven months postoperative.
Knee Motion: From 45 to 180 degrees, at seven months postoperative.
Remarks: Prophylactic penicillin. No infection developed. Nonapposition of fragments

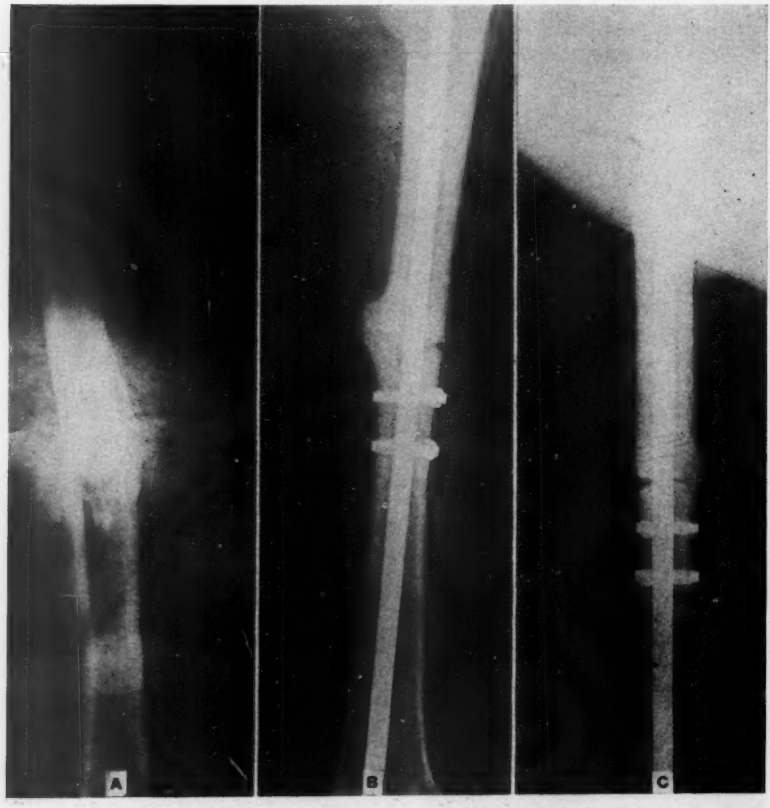

FIG. 5. Case 5. (a) A butterfly fracture of the mid-femoral shaft after six weeks in a plaster spica. (b and c) The same fracture nine months postoperative. Minor fragment fused. Callus organizing well. Fracture line still visible. Roentgenograms taken one month later show complete healing.

with skeletal traction. Operation approximately one month after soft tissue wound healed. Patient is working on limited status. Small fragments are seen to be completely fused with their parent fragments. Transverse fracture not united.

Case 5. (Fig. 5.) R. L. was a 43-year-old white man.
Fracture: *Butterfly* fracture of the upper midshaft, left femur.
Date of Operation: Sept. 15, 1951.
Weight-bearing: Limited full weight-bearing (standing), at end of two months. Unlimited weight-bearing, at end of three months.
Knee Motion: From 90 to 180 degrees, at nine months.
Remarks: Preoperative roentgenogram made six weeks after treatment in plaster spica. Ambulation depended on extremity containing femoral fracture, because of a badly comminuted fracture of the opposite tibia. One would have otherwise been tempted to treat the femur conservatively, in view of the callus seen. Patient carrying on full working activity from seventh month. Minor fragment united with its parent fragment after two months. Transverse fracture not completely united. (Most recent roentgenograms show complete healing.)

FIG. 6. Case 6. (a) A recent spiral oblique fracture of the lower third of the femoral shaft. (b and c) The same fracture seven and one-half months postoperative. Over abundant but well organized callus. Complete healing. Condyle not perforated by nail.

Case 6. (Fig. 6.) H. N. was a 41-year-old white man.

Fracture: Comminuted, spiral-oblique, fracture of the lower one-third of the left femur.

Date of Operation: Aug. 28, 1951.

Weight-bearing: Crutches with nonweight-bearing, one month postoperative. Partial weight-bearing, four months postoperative. Full weight-bearing, eight months postoperative.

Knee Motion: From 90 to 180 degrees, eight months postoperative.

Remarks: Active pulmonary tuberculosis. Attempt at removal of nail unsuccessful Nail cut off just above the greater trochanter.

Case 7. (Fig. 7.) C. O. was a 27-year-old white man.

Fractures: Bilateral markedly comminuted fractures of both femurs, lower middle and upper lower thirds.

Date of Operation: Aug. 13, 1951.

Remarks: Succumbed to irreversible shock on the eighth postoperative day while receiving preoperative enema. No demonstrable cause for death at autopsy. Fat emboli studies extensive.

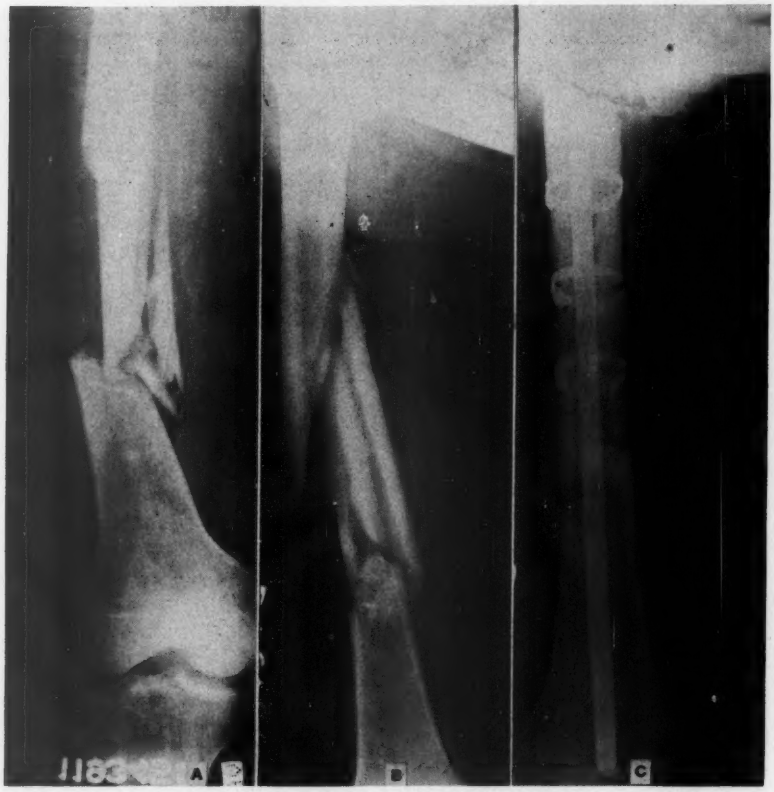

FIG. 7. Case 7. (a and b) A recent comminuted fracture of the lower middle and upper lower thirds of the right femoral shaft. (c) The same fracture immediately postoperative.

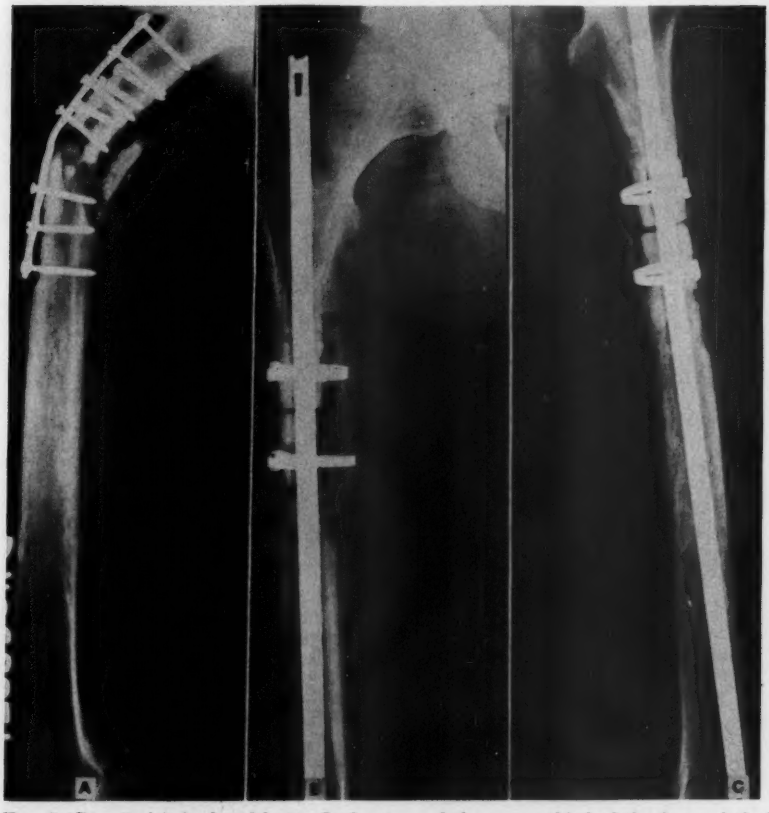

FIG. 8. Case 8. (a) A plated butterfly fracture of the upper third of the femoral shaft. Marked bending of the plate with the femur in traction (history). (b and c) The same fracture two months postoperative. Periosteal callus beginning to involve the cancellous graft. Mild to moderate osteoporosis.

Case 8. (Fig. 8.) R. T. was a 26-year-old white man.
Fracture: *Butterfly* fracture of the right femur with marked angulation, following plating and skeletal traction.
Date of Operation: Jan. 8, 1952.
Weight-bearing: Partial weight-bearing with crutches, one month postoperative.
Knee Motion: From 100 to 180 degrees, two months postoperative.
Remarks: If opposed transverse fragment surfaces had been much greater than one-fourth to one-third of shaft circumference, the onlay graft would have been used without bands or perhaps not used at all.

Case 9. (Fig. 9.) G. B. was a 24-year-old white man.
Fracture: Refracture of *butterfly* fracture of the right femur, four months after plating
Date of Operation: Nail and band, March 2, 1951.
Weight-bearing: Partial weight-bearing with crutches, at one month. Full weight-bearing, at four and one-half months.

FIG. 9. Case 9. (a) A butterfly fracture of the mid-femoral shaft prior to plating. (Plate broken four and one-half months after application.) (b and c) The same fracture nine and one-half months after nailing. Abundant callus incompletely organized. Healing not complete.

Knee Motion: From 75 to 180 degrees, nine and one-half months postoperative.
Remarks: Full activity undertaken from beginning of full weight-bearing. Healing believed not to be complete.

DISCUSSION

By far the greater number of femoral shaft fractures admitted to the Fracture-Orthopedic Service of the Dayton Veterans Hospital have been of the type for which internal fixation, in addition to intramedullary nailing, has been necessary in order to provide adequate stability for early ambulation.

Parham-Martin bands of the least ionic reactivity are mechanical agents which may be used for the adjunctive support of adult fractures in appropriate situations. The authors believe that their application should be governed by very clear-cut indications. The placing together of two metals in a wound is not acceptable if the modalities are appreciably ionically active as regards one another,

or as regards the bony tissue. This problem has been thoroughly studied by Venable and Stuck,[7] and commented upon by Key.[2] In the present cases, both the bands and nails have been composed of SMO (low carbon molybdenum) stainless steel.

The indications used in these cases, for supplementing the nails with bands, are predicated on the facts demonstrated by Böhler's cases, namely, that insufficiently supported nails will bend and that insufficiently supported fragments will permit angulation and/or shortening.[1] The complication of bending or breaking of the nail may well be minimized by the grade of steel available in this country. American made nails are said to contain fewer flaws than European nails.[5]

The experience gained from cases 1 and 2 indicates that stainless steel Parham-Martin bands, when used with intramedullary nails, are strong enough to overcome the marked tendency of the adductor muscles to produce varus[1] in sharply oblique fractures extending upward as far as the lower limit of the lesser trochanter or to within 7 or 8 centimeters of the tip of the greater trochanter. They are also strong enough to fix long oblique butterfly fragments (cases 5 and 9) and the fragments of some markedly comminuted fractures (cases 4 and 7).

SUMMARY AND CONCLUSIONS

Nine cases of open intramedullary nailing of femoral shaft fractures in adults are presented. Supplementary internal fixation has been necessary in each case.

SMO (low carbon molybdenum) stainless steel Parham-Martin bands were used as the supplementing agents, because they are believed to be the strongest and most practicable inert modalities available.

The types of femoral shaft fractures, in which internal fixation supplementary to intramedullary nailing is necessary, are illustrated and briefly discussed. The authors' concept of adjunctive internal support, for spiral oblique fractures and for indicated bone grafts, approaches that of Böhler. Böhler does not believe that long butterfly fragments are amenable to intramedullary nailing. To our knowledge, neither Böhler nor Street has illustrated a case of a sharply oblique fracture in the immediate subtrochanteric region, treated by intramedullary nailing.

Two complications occurred. One was a death due to unknown cause; the other, the failure to extract a nail after seven months.

A final report is planned, to cover all cases which can be followed.

On the basis of the material presented, it is believed at this writing that the use of adjunctive SMO (low carbon molybdenum) stainless steel Parham-Martin bands does not interfere with fracture healing. Their use appreciably widens the range of types of femoral shaft fractures, which can be successfully treated with SMO (low carbon molybdenum) stainless steel intramedullary nails.

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ROCCAL IN THE PROTECTION OF SURGICAL LINEN AS AN ASEPTIC BARRIER

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In a previous report,[1] we pointed out the fallacy of considering surgical linen as an aseptic barrier when it becomes wet. Nevertheless, until better materials are on hand, linen still will be required as an isolating medium to preserve the aseptic operative field. It, therefore, becomes obvious that some means must be used which will either prevent the wetting of the linen or will render it partially impervious to bacteria even when it has become wet. It is inconceivable at this time that the surgeon and the patient can be encased in a water impervious material. Skin respiration is necessary both for comfort as well as for health. Plastic materials are available, but these cannot be used to cover the entire patient nor the entire surgeon.

Accepting the thesis that a wet sheet or a wet surgical gown represents a contaminated area will do much to obviate this source of contamination. It is difficult to cover immediately any area which has become moistened and to discard all instruments which have come into contact with the wet material; or to replace any gowns which have become splashed with water or saline. In considering possible solutions to this problem, two main possibilities present themselves. One method would be to apply a water-repellant to the materials used. Another would be to impregnate them with a bactericidal substance which would not be affected by mere wetting and would, therefore, retain activity as an aseptic barrier.

We have made a series of experiments[2] by impregnating the surgical linen with Roccal* (10 per cent alkyl-dimethyl-benzyl-chloride) which is a technical grade of a quaternary ammonium bactericidal agent. We have found this chemical to be nonirritating to the skin, noncorrosive to instruments, and not injurious to the fabrics used in gowns and drapes. Furthermore, we have found that this chemical is in nowise altered by the usual methods of sterilization of linen equipment in an operating room. Some tests of the tensile strength of the fabric impregnated with the material have been made and have shown that there is no change at all.

A series of experiments were made with agar plate cultures of common skin bacteria and it was found that cloth treated with Roccal became an effective barrier even when moistened with sterile water. The tests were made by inoculating Petri dishes with a series of colonies of bacteria. These were covered with circles of treated and untreated fabric. All were moistened in approximately the same degree. Impressions were then made with the second series of agar

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* A product of Winthrop-Stearns, Inc.

plates and the second series was incubated. The resulting difference in the treated and untreated fabrics as barriers was very great. Actual colony counts showed a great reduction in the number of bacteria which were able to migrate through the treated fabrics.

When blood plasma and amniotic fluid were used as the wetting agent, there also was a great reduction in the migration of bacteria. It, however, appeared to be not quite as great as when physiologic sodium chloride solution or distilled water were used.

The logical site for the treating of these materials is the laundry. In this clinic surgical linen is subjected to 11 separate baths in the washing and rinsing process. The last of these is a neutral rinse at 110 F. In this rinse 100 gallons of water are used. To this quantity of water 1500 cc. of Roccal was added, which made a dilution of 1:2500. Tests showed that cloth rinsed in this solution contained approximately 0.44 milligrams of Roccal per gram of linen or 0.07 milligrams per square inch of the usual sheet.

Observations were then made in the operating room and colony counts were made with Roccal impregnated materials and ordinary linen. Cultures were taken from a large number of surgeons' gowns, Mayo stands, instrument tables, and wound drapes. In a consecutive series of 50 cultures taken on wet untreated material, 82 per cent showed bacterial growth. In series of 50 consecutive cultures taken of Roccal treated fabric, 14 per cent showed growth. This difference is significant. It is true that these studies were made by actually wetting areas in the operating room with sterile physiologic sodium chloride solution. They were not cultures taken of areas wet by accident, but were areas that were moistened deliberately so that comparable statistics could be obtained.

CONCLUSIONS

Surgical linens, gowns and wound drapes are not aseptic barriers when they have become wet.

Avoidance of any wetting, and considering any wet area on a surgical drape as a contaminated one is an ideal but impractical protection.

Significant differences in culture growths have been obtained in experimental studies of linen, both untreated and treated with Roccal.

The addition of Roccal to the laundry rinse is a simple and effective protection against the aqueous interruption of asepsis.

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UNUSUAL TUMORS OF THE FOREARM AND HAND

A REPORT ON MELORHEOSTOSIS AND NEUROFIBROMA

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The purpose of this presentation is to report and discuss data concerning some unusual tumors of the forearm and hand. Specifically, the tumors under consideration are: I, melorheostosis, a very rare syndrome, and II, an unusual neurofibroma of the median nerve, which we believe justifies a report at this time. Clinicians are generally familiar with neurofibroma, but there seems to be a considerable lack of knowledge and information among them in regard to melorheostosis. Since there is no apparent relationship between these two groups of tumors, each kind will be considered as an individual entity.

I. MELORHEOSTOSIS

(With Case Report)

Melorheostosis is a peculiar and rare form of osteosclerosis, which was first described by Léri and Joanny[8] in 1922. They noted an unusual degree of hyperostosis distributed on the side of a bone, comparable to and resembling the *tallow drippings on the side of a burning candle.* From the seemingly *flowing* nature of this hyperostotic condition, they created the name *melorheostosis,* a combination of the Greek terms for limb, flow and bone. Other synonyms for this condition are melorheostosis Léri, hyperostose en coulée de bougie, osteosis eburnisans monomelica and osteopathia hyperostotica congenita membria unius.

Since 1922, a total of 56 cases of melorheostosis has been reported. Only 24 of these have involved the upper extremities and a few of them have been limited only to the bones of the hand. In 1925, Lewin and MacLeod[9] reported the second case on record, which they, however, were unwilling to specifically classify. Excellent reviews on this subject have been written by Kraft,[7] Hall,[5] Spiegel and Koiransky,[17] and more recently by Wallensten.[18]

Etiology. The specific etiology of melorheostosis is obscure, although several theories as to its origin have been advanced. Léri[8] considered the possibility of mycotic or parasitic infection, but could not isolate any agent. Putti[14] advanced the theory of angiospasm, due to an imbalance within the autonomic nervous system. Lewin and MacLeod[9] suggested a neuropathic origin. Endocrine disturbances, with derangement of calcium metabolism, have been proposed by Michalowski[11] and by Fejer.[4] Moore and deLorimier[12] believe that the bone sclerosis is due to subperiosteal telangiectases, which give rise to hemorrhage in fetal life; and that osteosclerosis could represent a reaction following the hemor-

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rhages and the vascular changes. Zimmer[20] has suggested that the cause of melorheostosis is a disturbance in the development of the anlage bud of the part in question.

The 56 reported cases are no doubt a small group from which to draw definite conclusions. Sex is probably not a factor in this disease, since of the total number, 33 cases were males and 23 were females. In the 8 earliest case reports published, most observers believed this disease to be localized in one or another extremity. Bilateral changes were demonstrated in later cases reported by Bury,[1] Carpenter, Baker, Perry and Outland,[2] Sorel and Quenu,[16] and Fairbank.[3] Unilateral changes involving upper and lower extremities have been reported by Franklin and Matheson.[6] According to Müller-Alberti,[13] the disease has been known to occur simultaneously in the four extremities. This syndrome has been limited to involvement of vertebral bones in other cases, as reported by Woytek,[19] Sear[15] and Lievre.[10]

Pathology. Grossly, melorheostosis demonstrates a condition of osteosclerosis with irregularly shaped eburneous foci in the bone. The changes are seen in the epiphyses and along the diaphyses, usually extending along one side of the bone and resembling melted bone that has dripped and hardened, much like the wax from a burning candle. The sclerotic foci are partly in the endosteum and partly in the periosteum.

The new formation of bone progresses slowly, but in time may develop large masses which produce a visible tumor and also pain, resulting from the pressure on vessels and nerves. Finally, this large bone growth produces limitation of motion, due to tension of involved tissue as well as to joint involvement. Affected bones may be shortened but are almost never lengthened, and soft tissues in the neighboring vicinity of the diseased area may also be affected. Muscles may become atrophic and fibrotic, and connective tissue may become indurated and rigid.

Although there may be some question as to the specific cytologic changes in melorheostosis, there are these several common denominators in the cases thoroughly studied: The haversian system remains as a basic histologic pattern; fibrotic infiltration of the trabecular structure of the bone and fibrotic replacement of the marrow are regarded as the fundamental changes; and there is no evidence of any inflammatory process.

The interpretation of the condition of the osteoclasts and the osteoblasts has differed with many observers; the status of their activity apparently influences these varying opinions. Putti[14] describes a similar pathologic pattern but points out the presence of obliterated capillaries. These obliterated capillaries, he considers, may be a sign of ischemia, thus explaining the etiology of this disease.

Symptoms and Signs. The symptoms of melorheostosis entirely depend upon the location, the extent, and the activity of the pathologic changes. Since the osteosclerosis is focal in its pattern, there are no generalized constitutional manifestations of this disease. In general, we may say that its symptoms are pain and tumor, although a tumor may not be present in every case. Pain is the most common complaint and *is* present in almost every case. The degree of pain may vary

in given cases, but is rarely ever severe and almost never constant. In childhood the discomfort, in fact, is often transient. When the condition extends beyond the smooth confines of the periosteum, which is frequently the case, stony tumors and deformities may be not only present but will be obvious to the clinician. As the bony mass increases in size, there may be pressure against some of the regional nerves and involvement of muscle function. In some cases, there may be changes in the skin, with ulcerations due to stasis caused by pressure on the vessels and by coincidental scleroderma.

Diagnosis. It is apparent that melorheostosis has no specific symptoms or signs which are pathognomonic of this disease. The diagnosis can be established only by means of roentgenographic examination of the skeleton, which does give an entirely characteristic picture. The roentgenologist, familiar with this syndrome, should be able to verify the dense hyperostotic irregular, linear mass present apparently within the marrow. Rotation of the limb, with exposures taken in several planes, will reveal the margin of origin. At the onset of the disease, only the inner margin of the hyperostotic mass is irregular: at later stages, the process penetrates and distorts the smooth confining contour of the periosteum. The appearance of the local joint remains unchanged, even when the process is present on both opposing sides.

The replacement of structural reticulation by dense, stony opacity is specifically diagnostic. With its peculiar patchy linear distribution and selective localization to a single side of a bone, or to one of a pair of bones, this stony mass has the characteristic contour of a coarse, irregular wavy outline resembling the wax drippings down the side of a burning candle. This growth may be and *has* been confused with some of the other hyperostotic bone conditions. Among these are osseous syphilis osteosclerosis, osteopoikilosis, osteitis deformans, osteitis fibrosa, osteitis fibrosa disseminata, polyostotic fibrous dysplasia, myositis ossificans, osteogenic neoplasm or carcinomatosis, chronic osteomyelitis, traumatic or infectious ossifying periostitis, or nonspecific ossifying periostitis.

It should be pointed out here that other clinical laboratory tests have not been helpful in diagnosing this syndrome. To date, no changes resulting from melorheostosis have been established by hematologic observations of the blood, by urinalysis, and by blood chemistry studies.

Treatment. Melorheostosis has an insidious onset and usually is slowly progressive, with periods of temporary or permanent arrest but never with a reversal to normal bone. The disease tends to be chronic, although there is no evidence of resulting malignant degeneration. Pathologic fractures do not usually occur or at least have never been reported.

The treatment of this disease is purely symptomatic and consists of various types of physical therapy. Roentgen ray therapy has proved to be inefficacious. When joints become involved, orthopedic intervention may be indicated in order to relieve the static disturbances.

CASE REPORT

A. H., a 37-year-old Negro woman was admitted to the Cincinnati General Hospital in 1943 for a tumor involving the fourth and fifth digits of her right hand. She complained of

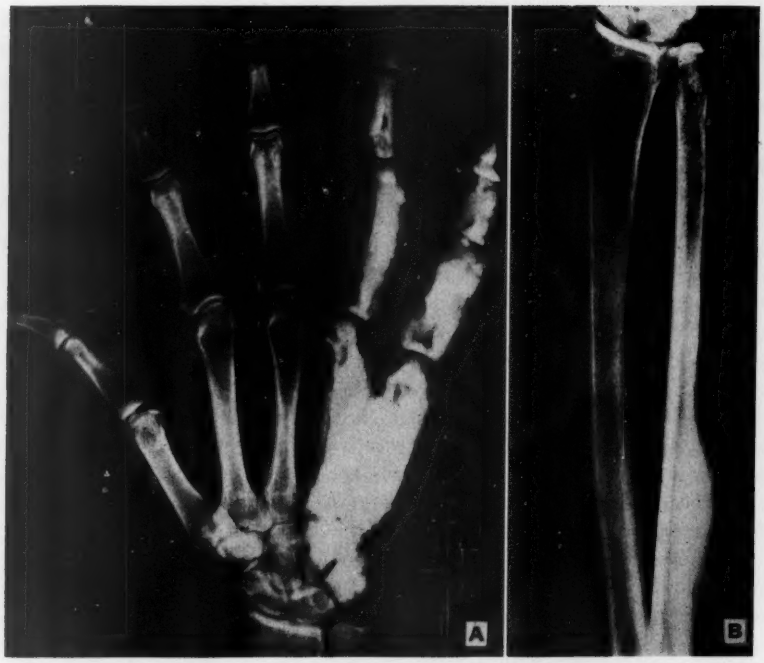

FIG. 1. *A*. Roentgenograms of the right hand showing involvement of the fourth and fifth metacarpal bones, the phalanges of these digits and the ulnar carpal region. *B*. The degree of osteosclerosis of the right ulnar bone is shown.

only slight discomfort in the involved region, but the presence of the tumor was of the utmost concern to her. A definite diagnosis was not made at that time.

The patient was first seen in the Hand Clinic on June 26, 1944, where her condition was followed for a period of eight years. At that time roentgenograms of her right hand established a diagnosis of melorheostosis, involving the fourth and fifth metacarpal bones and all of the phalanges of these two digits (Fig. 1). The metacarpal and interphalangeal joints were not involved by this sclerotic process. However, due to the presence of the tumor, the patient could not completely flex these two digits.

Except for obesity, her general physical examination was negative. Repeated tests for serologic reactions of the blood were also negative. All blood chemistry studies, including calcium–phosphorus ratio determinations, were within normal limits. Roentgenograms of the entire skeleton revealed no other bones to be involved.

In December 1946, roentgenograms of the right hand revealed some increase in density of the lesions already present. However, by March 1949, a survey and comparative films of both upper extremities demonstrated definite cortical thickening of the right ulna, comparable to the findings of the lesions seen in the fourth and fifth digits.

In January 1951, roentgenograms of the right forearm and hand showed that the condition involved the right ulna from the olecranon process to the middle third of the shaft. The process involved the ulnar carpal bones, and the entire contour of the fourth and fifth metacarpal bones, as well as the phalanges of the fourth and fifth fingers. Recent photographs (Fig. 2) demonstrate the effect of the lesion upon the function of the hand.

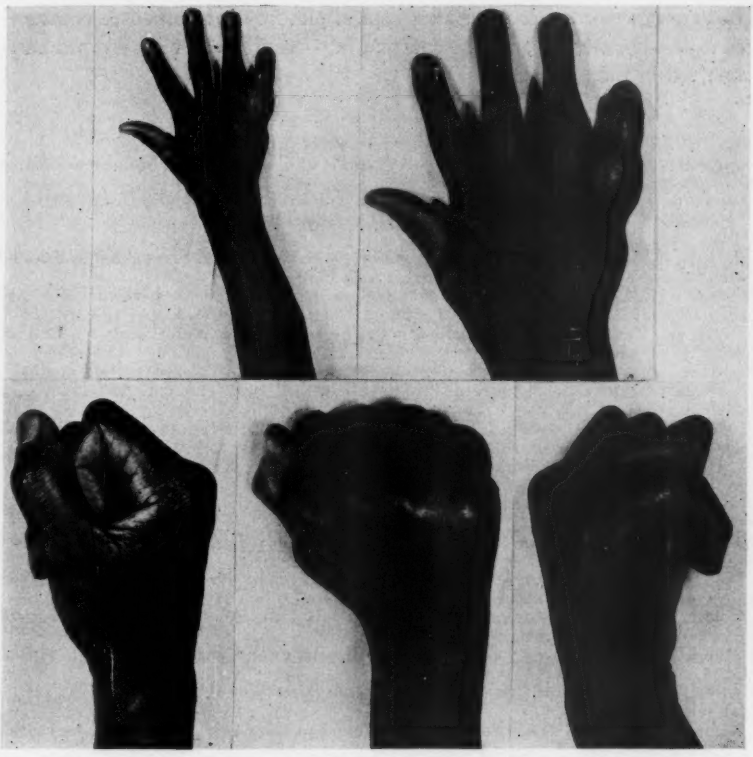

FIG. 2. Photographs demonstrating the visible tumor of the ulnar aspect of the right hand as well as the ability to flex and extend all of the digits. The ulnar lateral view demonstrates the limitation of flexion of the right little finger.

Comment: Insofar as can be determined, this patient represents the fifty-seventh case of melorheostosis reported in the published literature and is one belonging to the lesser groups involving, at first, the bones of the hand and, later, the forearm. Furthermore, this patient has been followed for eight years and there can be little doubt that her course demonstrates a slow, but progressive pattern of this disease. To this patient's knowledge, there is no familial history of this condition. By roentgenography, the specific diagnosis of melorheostosis Léri has been established in her case.

CONCLUSIONS

The syndrome of melorheostosis has been reviewed, with careful reference to its etiology, pathology, diagnosis and treatment. Although this condition is rare, clinicians should become familiar with its clinical and roentgenologic aspects in order to make the correct diagnosis.

The case described here presents a typical picture of melorheostosis involving bones in the right hand and forearm and is offered as the fifty-seventh case to be reported in the literature.

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UNUSUAL TUMORS OF THE FOREARM AND HAND

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II. NEUROFIBROMAS

(With Case Report)

In 1863, Virchow[16] divided primary tumors of the peripheral nerves into *true neuromas* and *false neuromas*. It is with the latter grouping that this discussion is primarily concerned.

Etiology. In the class of false neuromas, neurofibromas or pseudoneuromas arise from the connective tissue of nerves including the sheath of Schwann, and are more common than true neuromas. They may be single or multiple, benign or malignant.

The neurofibromas contain relatively few, if any, nerve fibers. They grow either within the nerve and infiltrate it, or rise from without and compress it. They attack not only the peripheral spinal nerves, but also their roots, especially those of the cauda equina and the cranial nerves, with special involvement of the acoustic and, less commonly, the trigeminal and optic nerves.

Since Virchow's contribution to the subject of peripheral nerve tumors, pathologists have been arguing over their correct classification. The chief divergences of opinion are concerned with the origin of the tumor and with the many factors involved in the relationships of the multiple tumors found in Von Recklinghausen's disease. Various authors have offered pathologic evidence to clear up this confused and complex subject. Excellent discussions on this condition are given in the reports of Penfield,[11, 12] Hosoi,[7] Stewart and Copeland,[13] Masson,[9, 10] Stout,[14, 15] Geschickter,[24] Bailey and Herrmann,[1] and Foot.[12]

In 1930, Lewis and Hart[8] reported 37 cases of pseudoneuromas (neurofibromas), among which were 3 cases involving the median nerves. This reference is of particular interest in connection with the present report, which is especially concerned with a neurofibroma involving the median nerve extending from the elbow into the hand.

Pathology. Since the origin of nerve fibromas is controversial, the pathologic picture is also controversial. Foot[3] has presented one of the best summaries of the problem that exists in the literature. He poses these questions asked by Lhermitte and Leroux concerning neurofibroma: "Does it contain Schwann cells or connective tissue or both? Is there such a neoplasm as neurofibroma of nerves? Is there such a neoplasm as primary sarcoma of nerves? Is neurosarcoma malignant neurofibroma?" Unequivocal answers to these questions have not been formulated, and thus the discussion continues.

Foot suggests a common sense approach to this controversy and says: "There seems to be no reason why there should not be such a neoplasm as pure fibroma of nerve, arising from endoneurium or perineurium, or, for that matter, epi-

neurium." His argument is that neurosarcoma appears to be a true fibrosarcoma in some cases, while in others, it has been shown to be a malignant neurilemmoma. These conditions could be just as likely primary in nerves as they could be primary in fascia or in any other connective tissue.

The greatest disagreement involving the pathology is concerned with the association to neurofibromatosis of other tumors in the clinical entity of neurofibroma. Stewart and Copeland[13] clearly have brought out the difficulties that beset any investigator trying to make a coherent theory that will explain neurofibromatosis or its association with tumors of the non-neurogenous group.

From the written material covering the pathology of neurofibroma and from the great discussion that goes on regarding neuroblastomatosis and the group of tumors commonly called *Von Recklinghausen's disease*, one may hope that this complicated picture will some day unfold and be clarified in the minds of all clinicians. Only improvement in staining technics and general agreement among pathologists regarding specific criteria seem to be needed for the correct diagnosing of neurofibroma. Perhaps, when this has been accomplished, we shall also know the connecting link between pure neurofibroma and melanoma, a moot point that has been discussed by Masson and others.

Symptoms and Signs. The symptomatology of neurofibroma is not striking, aside from the presence of a mass in the majority of instances. This is particularly true in the case of a neurofibroma of the forearm and hand. When a major nerve trunk is involved, there may be sensory disturbances. Especially present are pain and paresthesia, corresponding to the distribution of the affected fibers; however, these symptoms do not always occur. Motor disturbances are rare and do not result in more than a complaint of weakness in the affected part. This is true even when most, if not all, of the nerve fibers of a major nerve trunk are involved, and can be substantiated by case reports.

Diagnosis. The diagnosis of a neurofibroma is not an easy one clinically. As a matter of fact, the diagnosis often will not be made if there are no disturbances of nerve function. When the tumor lies beneath the skin and is movable except for one point of deep attachment, or is movable from side to side but not in the long axis of the course taken by nerves in the part involved, the possibility of attachment to nerve tissue is suggested. In its deeper manifestations, the neurofibroma cannot be differentiated from other benign tumors and cysts which may be found in the affected region, especially if it is inaccessible to palpation and there are no nerve manifestations.

The differential diagnosis in many cases includes the following tumors: Fibroma, lipoma, myxoma, sebaceous cyst, dermoid cyst, ganglion, giant-cell tumor, myosarcoma, lymphosarcoma and organized hematoma.

Treatment. The treatment of neurofibroma should demand some special consideration. When the tumor lies within an unimportant and small nerve, excision of the tumor with the nerve does not give rise to untoward results. On the other hand, when the tumor lies within an important major nerve trunk, very definite considerations arise and the management of the tumor often will tax the surgeon's ingenuity.

It may be said that in general, a neurofibroma should be removed. When the tumor has its origin within a major nerve trunk, it is frequently possible to remove the tumor including the capsule, without interfering with the majority of the nerve fibers of the nerve trunk. Under these circumstances, there may be a temporary impairment of function or a sensory disturbance; however, these symptoms usually disappear after a period of some months and leave no permanent damage, if the continuity of the whole nerve has been preserved. On the other hand, as will be shown by the case report here presented, the nerve trunk may be so involved over a long distance, that it is impossible to spare it in any degree. Under these circumstances, it is noteworthy that the distal functioning units do not show as much impairment in motor and sensory disturbances as one might expect. As a matter of fact, the functioning unit apparently compensates remarkably well during the period of tumor development, and the end result, after resection of a major portion of a nerve trunk, is not as striking as one would expect. Valuable contributions to the treatment of tumors of peripheral nerves have been made by Harrington,[5] Heuer,[6] and Cutler and Gross.[2]

As pointed out by Stout, neurofibromas must be removed in their entirety because of the frequent malignant changes which can occur if all tumor tissue has not been resected. On the other hand, he advocates that every effort be made to avoid unnecessary injury to important major nerve trunks, even at the risk of failing to remove all of the tumor.

Large neurofibromas, involving the ulnar and median nerves of the forearm and hand, pose specific problems to the surgeon who is interested in this anatomic site of the body. In our experience, we have had to deal with a very large neurofibroma of the median nerve of the left forearm and hand, and we believe that the surgical management of this case warrants its presentation.

CASE REPORT

A. G. C., a 25-year-old white woman, was admitted to the Christian R. Holmes Hospital on Aug. 22, 1945, complaining of a large tumor in her left forearm. This seemed to be associated with numbness and tingling in the left middle finger.

In 1933, the patient first noted the development of a small, tender nodule on the volar side of the left wrist, which gradually increased in size. In 1935, two other nodules were noted on the medial aspect of the elbow region; one in the upper forearm and one in the lower arm.

In 1937, the two volar nodules on the left wrist were removed in a Kentucky hospital. (The pathologic report of these two nodules could not be obtained.) After the operation, the patient noted numbness and tingling in the tips of her index and middle fingers. In 1943, she noted a cord-like tumor which extended from the left elbow region to the wrist which she described as a *knotty cord*. Since that time, the multiple tumor mass gradually increased in size and became more tender to touch and pressure.

Upon admission to the Holmes Hospital in 1945, this patient's general health was excellent. The entire physical examination was negative except for the left upper extremity, specifically the forearm and hand. This presented two large nodules about 3 centimeters in diameter, one in the upper third and the other in the middle third of the forearm. These nodules were tender to touch; seemed to be fixed in their deepest position, with the skin and subcutaneous tissue moving freely over them. On general percussion of the nodules, there was definite tingling in the tips of the left index and middle fingers.

Motor function of the left hand, and particularly of the thumb, was not disturbed. On sensory examination, the ulnar nerve was found to be intact. The only positive finding was definite hypesthesia of the areas of absolute median nerve sensory distribution. The blood picture was within normal limits and the urinalysis was negative.

On Aug. 27, 1945, under general anesthesia and with the use of a pneumatic cuff for a tourniquet, the tumor of the left forearm was explored through an incision extending from the lower portion of the left arm to the transverse volar ligament. This mass was found to be rising from the region of the left median nerve and was in contiguity with it, actually displacing the nerve bundles and fibers of the nerve. In order to remove this tumor, a median neurectomy was done, and the nerve was resected from a point at the elbow to the transverse volar ligament (Fig. 1).

The patient's postoperative convalescence was uneventful and the wound healed per

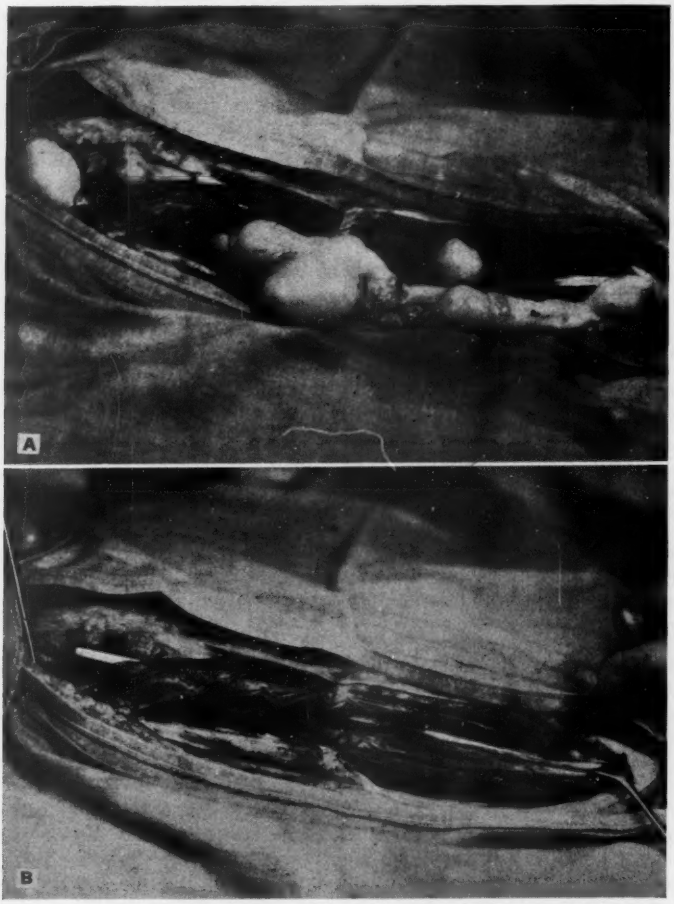

FIG. 1. *A*. Photograph of the gross appearance of the neurofibroma involving the median nerve in situ at the time of operation. *B*. The operative field after median neurectomy with a probe occupying the bed of the median nerve.

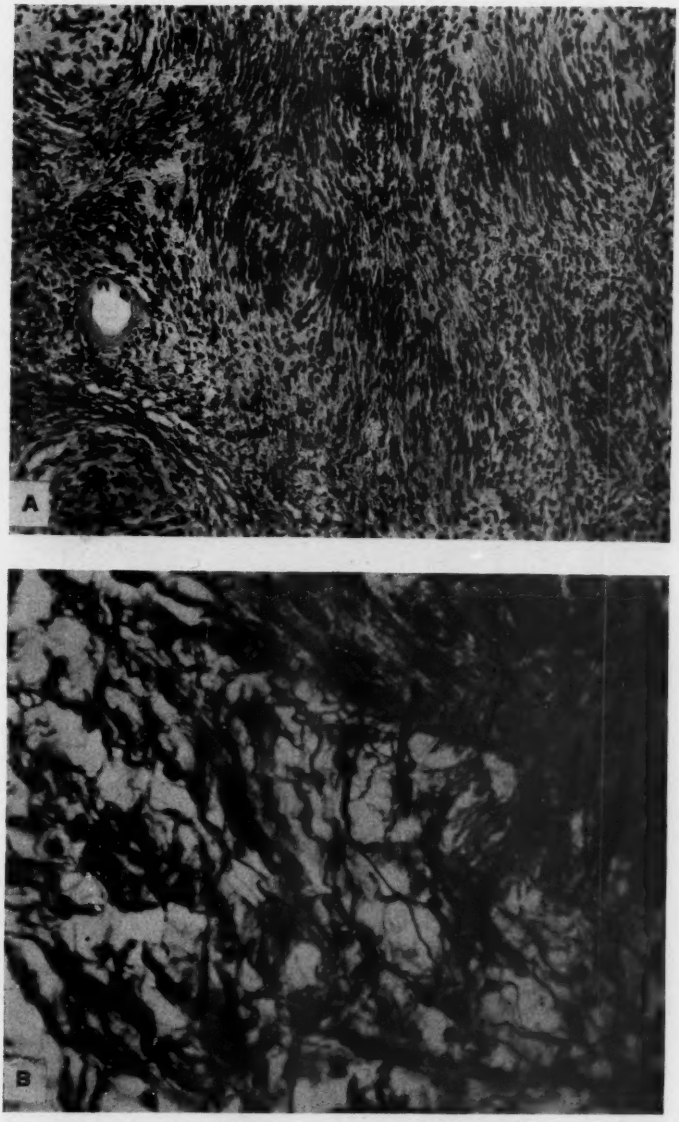

FIG. 2. *A*. Photomicrograph (haematoxylin and eosin stain of the tumor, ×160) demonstrating the general pattern and the tendency of palisading of the fibroblastic elements. *B*. Photomicrograph (Foot's modification of the Gross-Bielschowski stain of the tumor, ×900) demonstrating the background of reticulum and a branching nerve fiber.

FIG. 3. Photographs showing the ability of the patient to extend and flex all digits, rotate and oppose the thumb of the left hand.

primum. The specimen removed was carefully examined in the Neuropathology Laboratory at the Cincinnati General Hospital, and special sections were made and studied. The final diagnosis was neurofibroma (Fig. 2).

In the immediate postoperative period, the left hand demonstrated weakness of the intrinsic muscles of the thumb supplied by the median nerve. There was complete analgesia on the volar and dorsal aspects of the left index and middle fingers.

The patient was observed frequently and one year after operation her left hand showed atrophy of the abductor pollicus brevis, the opponens pollicus, and the flexor pollicus brevis muscles. There was suggestive atrophy of the first and second lumbrical muscles, and slight weakness of abduction of the thumb was noted, particularly on rotation of the thumb to a perpendicular position from the palm of the hand. Sensory examination revealed analgesia of the index and middle fingers and hypesthesia of the radial palmar aspect of the hand and thumb, and also hypesthesia of the radial side of the ring finger. The skin was in excellent condition and no trophic disturbances were noted. The skin of the left hand was cooler than that of the right hand.

This patient—a school teacher—continued her professional duties and used both of her hands extremely well. As one of the observers noted, there was a surprisingly complete degree of recovery in the function of the left hand, which may have been due to several factors: First, compensatory functional development as the median nerve neurofibroma slowly enlarged; second, unusual ulnar nerve overlap; and finally, excellent re-education of the involved parts by this patient.

In September 1945, a small subcutaneous tumor was removed from the right eyebrow region, which proved to be a neurofibroma. In 1949, two subcutaneous tumors were removed from the ulnar aspect of the left index finger and these also proved to be neurofibromas.

A survey of this patient's left forearm and hand, in January 1953 revealed small subcutaneous tumors, 3 mm. in size, on the ulnar aspect of the left index and middle fingers. Two larger tumors 6 mm. in size, were noted near the distal edge of the volar transverse ligament. The left hand represented an excellent functioning unit (Fig. 3). Unquestionably, a casual observation would miss any impairment of motor or sensory function in this hand.

Comment: This patient presented multiple neurofibromas, limited chiefly to the left forearm and hand. Most prominent was the large multinodular neurofibroma which involved the median nerve from the elbow to the transverse carpal ligament in the left forearm. Resection of this tumor was made by median neurectomy, which created a deficient span of approximately 20 cm. between the proximal and distal ends of the nerve.

We would like to re-emphasize the final result in this patient following median neurectomy. The left hand again has become a surprisingly excellent functioning unit, and the explanation for this is based upon three main factors: First, unusual compensatory functional development during the period of progressive enlargement of the tumor; second, questionable ulnar nerve overlap and, finally, re-education of the involved parts by an extremely intelligent patient.

CONCLUSIONS

The general aspects of neurofibroma have been discussed, and divergent opinions regarding the pathologic diagnosis have been presented. The treatment of neurofibroma involving major nerve trunks has been stressed with the particular point that good surgical judgment and ingenuity are important factors in its treatment in any location whatever, particularly, those arising in the hand and forearm.

An unusually large neurofibroma arising in the median nerve of the left forearm has been presented. In this case median neurectomy was necessarily done seven years ago. The final result has been discussed and reasons have been advanced for the excellent functioning left hand following major neurectomy.

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ADRENAL INSUFFICIENCY IN THERMAL BURN WITH SEPTICEMIA

SUCCESSFUL REPLACEMENT THERAPY

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With the introduction of sulfonamide drugs, antibiotics, and adrenal cortical hormone preparations, more and more patients who have acute adrenal insufficiency (Waterhouse-Friderichsen Syndrome)[8] are recovering from a condition universally fatal prior to 1940.[3] Moreover, this clinical syndrome is now being recognized with increasing frequency as a complication of other diseases and conditions. Although once considered to be due almost solely to adrenal hemorrhage secondary to meningococcal septicemia, the etiologic agents which can produce this same picture are now thought to be numerous. A relatively sudden decrease in adrenal cortical function may occur during pregnancy,[2] as a result of overwhelming septicemia, or from thrombo-embolic phenomena such as in sickle cell disease.[7] It may also produce signs of an acute abdominal crisis.[1] Petechiae and purpuric lesions may or may not be present.[2]

The case here described, of acute adrenal insufficiency due to a massive septicemia following an intravenous infusion, was successfully treated with cortisone, aqueous adrenal cortical extract, and antibiotics. It is believed that sufficient adrenal cortical replacement therapy in this patient was responsible for his survival.

CASE REPORT

W. W., a 14-year-white male was admitted to the plastic surgery service, John Gaston Hospital, Memphis, Tennessee, at 8:15 p.m. on Dec. 28, 1952. Shortly before admission he had received burns of both legs when he inadvertently overturned a container of burning gasoline. The burned areas—principally second and third degree—comprised approximately 15 per cent of his total body surface area.

Physical examination at time of admission was essentially negative except for the burns on both legs. His temperature was 98.6 F., pulse 96, respiration 24, and blood pressure 140/80. After the legs had been gently debrided and dressed, 1000 cc. of 5 per cent glucose in physiologic sodium chloride solution was started intravenously while blood was being typed and crossmatched. At 9:00 p.m. 500 cc. of plasma was given intravenously without apparent reaction. At 10:00 p.m. he was transfused with 500 cc. of whole blood, following the completion of which he experienced a severe chill accompanied by profuse vomiting. The blood pressure which had been 140/80 at the time of admission dropped to 70/0, the pulse rate accelerated to 120 beats per minute, and the body temperature rose to 106.4 F. Cyanosis was noted before the end of 24 hours, and the blood pressure remained below 90/60, except for infrequent and sporadic rises from 90/60 to 120/80 for brief periods, for the next 36 hours. During this period he was semicomatose and extremely restless. The rectal temperature ranged from 103 to 105 F. At the end of 40 hours following the burn, widespread petechiae, a massive left subconjunctival hemorrhage, and multiple ecchy-

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TABLE I

Laboratory data

Date (1952)	Hematocrit	CO_2CP Vol. %	Chlorides mEq/l	NPN	Creatinine	Eosinophil Count	Serum Bilirubin	Plasma Na + mEq/l	Plasma K + mEq/l	Urine
12/28	38									
12/29										
12/30	45	36	99	91				143.8	5.1	No cells present
12/31		48		108	2.8		1 min. 0.7; total 5.6			10–12 red blood cells
1/1		67	103	81	2.0	28		139	3.8	
1/2	44	74	100	66		2		148	3.9	
1/3										
1/4				40						
1/5	47	50	115	33		8	1 min. 0.4; total 2.4	144	4.4	
1/6	45	52	97	37		0		143	4.4	Neg.
1/7		65	99	31	1.0					Neg.
1/8										Neg.
1/9	46					0		142	5.3	
1/10		50	96	42						

motic areas were noted at hypodermic injection sites. Blood studies at this time showed thrombocytopenia, prolonged coagulation and bleeding times, and defective clot retraction. A blood culture at this time was positive for hemolytic staphylococcus aureus. Other laboratory data are listed in table I.

Intravenous fluids and blood were given in the quantities necessary for both treatment of the burns and to combat persistent hypotension. At the end of 60 hours he had received 11,500 cc. of intravenous fluids and had only a scant urinary output. His abdomen was much distended, necessitating nasogastric suction. Large quantities of bright red blood were aspirated from the stomach.[4] In addition, generalized venous engorgement was noted, accompanied by rapid respirations, indicating pulmonary congestive changes. The diagnosis of acute adrenal insufficiency having been made, cortisone and aqueous adrenal cortical extract were administered. Eight hours after having received 100 mg. of cortisone intramuscularly and two doses of 50 cc. each of aqueous cortical extract intravenously, the patient regained consciousness and appeared to be much improved. The blood pressure stabilized around 112/80 and diuresis began. He received four 50 cc. intravenous injections of aqueous adrenal cortical extract within 24 hours. Cortisone was continued for 10 days. The total dose of cortisone was 1.075 gm. In addition to hormone therapy he received one million units of aqueous penicillin intramuscularly every three hours and 0.5 grams of dihydrostreptomycin intramuscularly every six hours.

His third degree burn areas were covered with split thickness autogenous grafts on his twenty-fifth hospital day, and he was discharged on the thirty-seventh hospital day, completely healed and asymptomatic.

DISCUSSION

Since it definitely cannot be ascertained clinically at which point damage already sustained by the adrenal glands is reversible, prompt, vigorous, and aggressive therapy must be instituted as soon as the diagnosis of adrenal cortical insufficiency is suspected. Until a more satisfactory method of treatment is de-

vised, it seems rational to use all of the available adrenal cortical hormone preparations in large quantities as substitution therapy in addition to massive doses of chemotherapeutic agents and intensive general supportive care. Cortisone appears at present to be the hormonal agent of choice, but the fulminating nature of the syndrome warrants broad coverage with other preparations.[5, 9] Recent reports indicate that cortisone produces dramatic changes.[6, 7] This case confirms these reports.

As previously mentioned, the damage already sustained by the adrenals is not always reflected in the clinical picture at the time the diagnosis is made. For this reason in no case of acute adrenal insufficiency can a favorable prognosis be made early. This case presentation with recovery is not meant to purport that all cases of acute adrenal insufficiency will recover on any regimen, but if there is to be any hope for recovery the diagnosis should be made early, and vigorous therapy instituted.

SUMMARY

The recovery of case of acute adrenal insufficiency due to septicemia following transfusion is reported.

Acute adrenal insufficiency is being recognized more frequently as a complication of other diseases and conditions.

Cortisone was considered to have produced dramatic improvement in this case.

The adrenal cortical hormones, especially cortisone, may serve as replacement therapy for adrenal cortical insufficiency until there has been recovery or regeneration and the gland has resumed its normal function.

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PRIMARY THROMBOSIS OF THE ABDOMINAL AORTA ASSOCIATED WITH PRIMARY THROMBOSIS OF THE LEFT PULMONARY ARTERY

REPORT OF A CASE

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This case report is presented partly because of the relative rarity of primary thrombosis of the abdominal aorta, and also becuase of the complicating finding at autopsy of primary thrombosis of the left pulmonary artery. So far as could be ascertained from a review of the literature, no such combination of findings in a case has been reported.

CASE HISTORY

W. F. S., white man, aged 62, was admitted to the Wadsworth Veterans Administration Hospital on June 10, 1952 with an admission diagnosis of peripheral vascular disease of the left leg.

Present Illness. The patient was struck by a heavy oil can on the back of the left leg just below the knee in March 1952. Following the accident, the leg became blue and swollen, and gradually healed. At about this time, he noted the onset of weakness in both legs and a numbness and coldness of the left leg which was aggravated by walking. Walking caused pain in the left leg and thigh. Also, about this same time, he noted the onset of increasing difficulty in maintaining erection, and sexual relationship was rendered impossible.

The past history and review of systems were negative and noncontributory.

Physical examination. The significant findings were as follows: lungs, clear to percussion and auscultation; heart, within normal limits; vascular, minimal sclerosis of the peripheral vessels. Extremities, moderate generalized atrophy of both lower extremities; left leg showed rubor in dependent position and blanching on elevation; left foot cold and no pulse was felt in either the dorsalis pedis or the posterior tibial arteries; neurologic examination was within normal limits.

The laboratory findings were all within normal limits on admission.

Hospital Course. This was uneventful until the evening of June 16. At about 6 p.m. he had a sudden onset of severe pain in the low back and in both legs and increasing weakness of both legs. The pain and weakness progressed and soon both legs became numb, the pain was mainly in the lower back. Examination at that time showed both legs to be blanched and cold, with some rubor on being placed in a dependent position. No arterial pulses were palpable below the level of the abdominal aorta at the umbilicus and the lower extremity reflexes were bilaterally absent. A diagnosis of Leriche's Syndrome with more recent sudden occlusion of the abdominal aorta by an embolus was made and operation was advised.

At operation, on opening the abdominal aorta at a point just above its bifurcation, an old organized thrombus which almost completely occluded the vessel was found. Superimposed upon this was a recent red thrombus which had complete shut off the already narrowed channel left in the vessel. Most of the old thrombus and all of the recent red thrombus were removed and the incision in the vessel was closed. Pulsation of both femoral

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The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

arteries, which had been absent, returned. The patient was returned to the ward. The postoperative course was unsatisfactory. He developed increasing cyanosis of the left lower leg which gradually became more definitely demarcated. He developed uremia from which he died on June 20, 1952.

Autopsy Report. The pertinent findings were as follows:

External examination reveals an unhealed, midline abdominal incision. There is bluish mottling of the lower two-thirds of the left leg.

On opening the abdomen there is noted bilaterally extreme reddish discoloration of the retroperitoneal tissues in the area of the psoas muscles. The pleural cavity, lungs, heart and pericardium appear normal.

The lower lobe of the right lung shows a general increase in density and, on section, this lobe presents numerous areas of increased density which are greyish in color and extrude pus on pressure. The left lung has a firm, depressed area, 10 cm. in diameter, in the lateral aspect of the lower lobe. On section at the hilum of the left lung, the main pulmonary artery is found to be completely occluded by grey-pink thrombus. In some areas, this thrombus appears to be firmly adherent to the vessel wall throughout its circumference. Sections of the upper left lobe show it to be red and moist; sections through the lower lobe reveal it to be red, firm and relatively dry. The smaller arteries throughout the lower lobe contain thrombi.

The heart is essentially normal.

On inspection of the abdominal aorta, there is noted an incision 2.5 cm. in length in its anterior wall superior to the bifurcation. This incision is closed by a running silk suture and presents on its surface clotted blood. On opening the aorta, a grey-pink thrombus is found which extends from a point just below the origin of the superior mesenteric artery to about 3 cm. below the origin of the inferior mesenteric artery. It is firmly adherent to the wall of the aorta throughout its length. It partially occludes the origin of the left renal artery and completely occludes the origin of the inferior mesenteric artery. Sections through the aorta show a small dissecting aneurysm beginning at the level of the interior mesenteric artery and extending 4.5 cm. downward along the left lateral wall of the artery. The inferior mesenteric artery is completely occluded by a thrombus extending 5 cm. downward from its origin. The common iliacs appear to be free from thrombi, but at the bifurcation of each appears a thrombus which completely occludes the hypogastric and external iliac vessels on the left and partially occludes them on the right. Diligent search of the deep veins of the lower extremities does not show any evidence of thrombophlebitis.

The right kidney is normal. In the lower pole of the left kidney there are several wedge-shaped, yellowish areas in the cortex; each surrounded by an area showing petechial hemorrhage.

The remainder of the gross pathologic findings are within normal limits except for evidence of cerebral cortical atrophy and the presence of hemorrhagic areas in the small bowel.

Microscopic examination. In the lungs in some areas, there is dilatation and rupture of the alveolar spaces. These spaces in other areas, are filled with pus and blood, and some are beginning to show necrotic change. In other areas, branches of the pulmonary arteries are occluded by thrombi made up of red cells, fibrin, plasma cells and lymphocytes. The intimal surface of these arteries, is, in a great many areas, completely obscured incident to the organization of the thrombus.

Sections of the aorta show a sclerotic change involving the intima and, in some areas, the media as well. These sclerotic plaques contain cholesterol and calcific deposits. Superimposed upon the sclerotic process is a thrombus of red cells, fibrin, scattered plasma cells and lymphocytes. This only partially occludes the vessel and, in some areas, shows evidence of organization and attempts at recanalization. No particular degree of periarteritis is noted.

In the small intestine there is a fairly generalized inflammatory change involving chiefly the subserosa but, in other areas, extending into the submucosa.

DISCUSSION

The syndrome characterized by weakness and intermittent claudication in the lower extremities; global atrophy of the legs, and loss of the sexual power of erection was first clearly described by Leriche in 1923.[8] A later report by the same author bears out the relationship between the pathology of slow thrombosis of the abdominal aorta and the symptom complex described by him. As noted by Greenfield,[5] the condition was first described pathologically by Graham in 1814. Since then, numerous cases have been reported, and Greenfield points out that evidence has been accumulating to indicate that the real incidence of the condition is higher than the number of case reports would indicate.

The mean autopsy incidence of the condition has been calculated to be 0.12 per cent,[10] but, as already stated, the actual incidence well may be higher. Other reports[3, 4, 6, 9, 14] serve to substantiate the truth of this assertion. In all of these reports it is emphasized that if one has the condition in mind, the diagnosis is relatively easy. With a history as outlined above, it might be said that the one physical finding of absent pulsations below the level of the umbilicus would be sufficient to make the diagnosis at least probable. Other authors[2, 11] cite cases in which the diagnosis was first made clinically, and later confirmed radiographically by retrograde visualization of the abdominal aorta.

As regards pathogenesis, the case presented here certainly falls into the category of the development of a primary thrombus on an underlying ulcerated atheromatous plaque.[11, 14] It also seems certain that this patient, when first admitted, had incomplete occlusion of the abdominal aorta and that the episode which occurred while he was in the hospital, and for which he was operated upon, was brought on by a complete obstruction of an already markedly narrowed vessel. Whether this obstruction was due to further thrombus formation or to embolism is difficult to say as the specimen removed at the time of operation unfortunately was not saved for pathologic examination.

The treatment for the condition ordinarily advocated is lumbar sympathectomy[2] with or without added resection of the involved portion of the aorta as advised by Leriche.[8] In this particular instance, however, any treatment probably would have been doomed to failure because of the extent of the pathology as well as the added insult of complete and sudden obstruction of the already almost completely blocked abdominal aorta.

The coincidental finding at autopsy of primary thrombosis of the left pulmonary artery might be said to add a possibility for speculation on the causes of both of these relatively rare conditions. Brenner,[1] in a masterful monograph on the subject "The Pathology of the Vessels of the Pulmonary Circulation", concludes that the majority of reported cases of primary thrombosis of the pulmonary artery are, in reality, cases of embolic obstruction of the smaller branches with later superimposed retrograde thrombosis. No evidences of thrombophlebitis were found in this case. Patterson[12] presents 2 cases in which the apparent cause of the thrombosis of the pulmonary arteries was a rupture of the capillary vessels in the wall of the artery resulting in intimal hemorrhage and thus, in turn, giving rise to a nidus for thrombus formation. Savacool and Charr,[13] in a report of 100 cases

of thrombosis of the pulmonary artery in tuberculous patients give evidence which indicates that pulmonary disease also can cause retrograde thrombosis of the pulmonary artery, as all of their cases showed thrombus formation beginning in the smaller branches of the pulmonary vessels immediately adjacent to the involved lung. Irwin[7] presents a case of massive thrombosis of both main branches of the pulmonary artery based on the presence of multiple congenital aneurysms and band formation in the pulmonary artery. His case also was one in which the pulmonary circulation was embarrassed enough to cause chronic cor pulmonale. Brenner points out, that from 61 to 86 per cent of the cross section of the pulmonary artery has to be blocked before there is any demonstrable effect on the lesser circulation. This fact is, of course, taken advantage of in modern chest surgery and also accounts for the fact that this patient had no symptoms referable to the pulmonary system.

Just what connection there was between the development of the thrombosis of the abdominal aorta and also in the left pulmonary artery is difficult to say. Some have attributed the cause for thrombus development to some factor in the blood. Sclerosis of the vessel walls has also been considered a cause and, this was a likely factor in the development of the thrombosis of the abdominal aorta. In this connection it has also been shown by Brenner[1] that sclerosis of the pulmonary arterial system is far more common than is usually thought. Although, in this case, definite sclerosis of the walls of the pulmonary arteries was not mentioned, the fact that the anatomy of the vessel wall was completely obscured, incident to the organization of the thrombus, leads one to the conclusion that sclerosis was present. Why only the left lung was involved is difficult to say, particularly in view of the fact that, according to reported cases in diseased lungs,[13] thrombosis is far more common in the right lung.

SUMMARY

A report of a case of primary thrombosis of the abdominal aorta, associated with primary thrombosis of the left pulmonary artery is presented. The first diagnosis was made preoperatively and proved correct at operation. The second diagnosis was unsuspected during life and discovered only at autopsy. A brief discussion of various factors related to both conditions is given together with a surmise as to the possible relationship between them.

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THE NATURAL HISTORY OF OSTEOCARTILAGINOUS EXOSTOSES (OSTEOCHONDROMA)

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The most common benign tumor of bone is the osteocartilaginous exostosis or osteochondroma.[5] It may occur in any bone, but is most common in the long bones and predominantly in the metaphyseal regions. The usual locations are the distal metaphysis of the femur and the proximal metaphysis of the tibia. The lesion may be solitary or multiple. The latter is generally believed to represent a dominant hereditary trait. There is no pathologic difference between the solitary and the multiple lesions—each representing a developmental skeletal anomaly.[1] These growths present characteristics of neoplasms; may undergo malignant degeneration and cause death by widespread metastases. Chrondrosarcoma—the malignant aspect of the disease—frequently contains myxoid and fibrous elements and typically disseminates by invasion of venous channels. This sarcoma has its origin in the growing elements of the cartilaginous cap of the exostosis.

Whether solitary or multiple, the osteochondroma is a cartilaginous capped bony projection from the surface of the affected bone. It is never found arising from the marrow surface of a bone. At its site of attachment there is a cortical defect and the mass of the exostosis merges with the spongy portion of the parent bone. The basis of the exostosis apparently stems from perverted activity of the periosteum which shows a tendency to form aberrant cartilaginous islands. These islands in turn are abnormal in their growth patterns, both in chronologic and histologic properties. They continue to grow and to undergo endochondral ossification, forming the bony mass of the exostosis. Uncontrolled growth of these cartilaginous islands forms the anlage of the chondrosarcoma.[4]

Kieth has pointed out that most cases of multiple hereditary exostoses show a defective molding of bone with resultant broadening and blunting of the affected metaphyseal regions.[3] Lichtenstein, in reviewing 200 cases of exostoses, both solitary and multiple, reports that there is no significant sex difference in occurrence of the growths. The greatest number of exostoses manifest themselves in childhood or adolescence, but have been reported as occurring in patients between the ages of 10 months and 85 years. In his series, 80 per cent of patients operated upon were under 21 years of age.[2]

The clinical complaints due to exostosis reflect its slow growth and freedom from serious disability. There is usually a history of a prolonged period between recognition and the time surgical attention is sought. Occasionally an osteochondroma forms a primarily painful tumor by virtue of its rapid expansion and tension upon the periosteum. The stalk of an exostosis is a frequent site of fracture from trauma. The most common complaints are made for cosmetic reasons or because of painful pressure areas, such as pressure within shoes. The potential and actual interference with pregnancy is not unusual in the cases of endopelvic osteochondromas in women.

The osteochrondroma is a sessile or stalked bony projection of variable size and contour extending from the cortex of the affected bone. It may be smooth and gradually unite with the shaft of contiguous bone or arise abruptly and present an irregular knobby or cauliflower bony mass. Some of the more tubular exostoses may have spiked ends. Osteochondromas appear in a wide variety of sizes from less than 1.0 cm. to greater than 30 cm. across at their greatest diameter.

The exostosis is covered with periosteum which is continuous with the periosteum of the adjacent affected bone. The periosteum of the osteochondroma differs from the normal periosteum in that it strips from the tumor with difficulty and is composed of irregularly thickened, avascular, fibrous tissue in indefinite layers. The surface underlying the periosteum is bluish-white hyaline cartilage. In general, the younger the patient the more pronounced is this cartilaginous cap. The cap may vary in thickness from less than 1.0 mm. to over 1.0 cm., and its contiguous border, if actively growing, is an area of endochondral ossification. The area of ossification presents a yellow brown plate underlying the spongy bone that composes the mass of the exostosis. The marrow cavity is usually filled with fatty marrow, but may contain myeloid elements.

Microscopic sections of a growing cartilaginous cap show areas of proliferating cartilaginous cells in the deeper layers much like articular cartilage. As suggested by its terminology, the exostosis grows by endochondral ossification in a manner similar to that which occurs at the epiphyseal plate of growing bone. There is a frequent tendency of absorption of cartilaginous cores in the underlying subchondral trabeculae of the adjacent bone. In other cases the cartilaginous cores persist and produce areas of mottled radiolucency on the roentgenogram. When this resolution is faulty, the area may be replaced with fibrous marrow, calcified bodies or debris, and occasionally myxoid elements.

The age at which an exostosis ceases to grow varies, but generally coincides with the end of the growth period of the person. Patients in whom *growth halt* precedes or is subsequent to normal growth by many years are not unusual. In a young adult, the cartilaginous cap is apt to show areas of quiescent cartilage exhibiting calcification or other regressive changes. In older patients, the cap tends to show extensive regressive changes with varying degrees of involution, although remnants of irregularly active cartilage may persist in the sixth and seventh decades. These residual cartilaginous nests, although usually inactive, have the capacity to reactivate and form the basis of chondrosarcoma that develops later in life. The exostoses which are situated so that they irritate overlying muscle and tendons frequently separate themselves from these structures with bursae. A bursa may be the site of a painful inflammatory process or may form a tender, separated, calcified mass.[6]

The first reported case of sarcoma developing in an osteochondroma was published by Weber in 1866, and frequent attention has been called to the subject in medical literature since, although its occurrence is rare enough to merit report of unusual isolated cases.[7] In some cases the new growth is first noted after a traumatic or adjacent inflammatory process. The evolution of malignant change is usually a slow and insidious process, requiring months or years for development, with the tumor attaining great size. On the contrary, the chondrosarcoma may

grow rapidly to large proportions; break into venous channels; disseminate and cause death in a few months from the time it is first noticed. The usual metastatic sites are the lung, brain and the myocardium. This rapid fatal course is unusual. The tumor, more commonly, remains locally invasive for years without distant spread. It is usual for death to intervene and chondrosarcoma be an incidental finding at autopsy.

Roentgenologically chondrosarcomas tend to show mottled calcified areas; very dense in many cases.

The pathologist when studying biopsy tissue or reviewing an excised mass may consider an osteochondroma benign. The sole clues to malignancy are atypical cartilaginous cells in the proliferating areas. The surgeon, when removing tissue for biopsy, can miss malignant areas.

The treatment of an exostosis and the complicating chondrosarcoma will frequently tax surgical judgment. The mere presence of an exostosis is not indication for its removal, unless it presents a cosmetic or pressure problem. As a general rule, any exostosis that takes on rapid growth should be considered to be a chondrosarcoma and should be treated accordingly. The exostosis should be widely excised, including a generous section of adjacent periosteum. Recurrence may develop in the remaining areas of perverted periosteum, which was the basis of the original tumor.

Local wide resection may be adequate treatment for small osteochondrosarcomas. Such cases should be observed carefully and roentgenograms should be made frequently. At the first sign of recurrence, amputation—one joint proximal to the growth—is indicated as the tumor typically will spread up the marrow cavity to the diaphyseal plate. Many surgeons prefer primary amputation in all cases in which the excised tumor is reported to be chondrosarcoma.

The treatment of large, progressively growing tumors with mixed tissue elements should be treated by amputation if the site permits. Local resection is reserved for the occasional patient who refuses amputation. Resection of chondrosarcomas of the pelvis and shoulder girdle is difficult. Only occasionally are they amenable to hemipelvectomy or radical removal of the shoulder girdle. Roentgenotherapy has no place in the treatment of chondrosarcoma because the tumor is highly radioresistant. Implantation of radon seeds or attempts at tumor localization with radioisotopes fail for the same reason. To date there are no palliative measures to abate the malignancy after it is widespread.

The incidence of chrondrosarcoma in solitary exostoses is low, occurring in from 1 to 3 per cent. Preventive excision of exostoses hardly seems justifiable. Regular roentgenograms and prompt extirpation upon evidence of increasing growth is the rule. The incidence of chondrosarcoma in multiple hereditary exostoses is much higher; ranging up to 11 per cent. In this group close regular study with roentgenograms is imperative.

CASE HISTORY

Mrs. M. S. was a 54-year-old widowed housewife who had not been gainfully employed for 11 years. Prior to that time she had worked as a steam presser and ironer in a laundry for many years. Her first complaints pertinent to the present disability began 13 years ago

when she accidentally fell down five steps and sustained bruises resulting in swelling of both knees and left hip. Roentgenograms were negative for fracture or dislocation but it was observed that an osteochondroma 2 by 3 by 3 cm. protruded from the upper posterior aspect of the left tibia. A similar lesion 2 by 2 by 2 cm. was present at the same site on the right tibia and multiple smaller osteochondromas were found over posterior aspects of both femurs and pelvic inlet. She recovered from her injuries and returned to work. For two years she had no complaint.

In early 1942, she was examined after complaints of tumor and pain in the upper left leg. She then had a stony hard mass—immovable and nontender, measuring 4 by 6 by 6 cm. and presenting a bossed surface—protruding from the upper posterior end of the left tibia. Roentgenogram showed that the previous osteochondroma—consisting of a cone of cancellous bone—had changed into a cauliflower like mass with irregularly mottled areas of calcification with virtual replacement of the cone. There was no joint involvement. Excision was recommended but she refused because she feared amputation might result.

Two years later she was dependent upon codiene in small doses for relief of pain. The tumor had increased approximately 100 per cent in size; still did not involve the knee joint, but made it impossible to flex the knee more than 40 per cent of normal. She submitted to biopsy section of the tumor at this time and again local excision was recommended but she refused for fear of amputation. The pathologic report was "chondroma with degeneration and calcification." Histologic sections or specimens of the biopsy were not available for comparison with the current studies. The patient was not seen for five years, during which time she had a hysterectomy for menopausal bleeding.

She returned again in the spring of 1949 with complete disability from pain and weakness in the left leg. She walked with a cane and drug her left foot most of the time, using it for support as she rested. The tumor had grown and how measured 20 by 20 by 25 cm. It presented the same mottled appearance by roentgenogram with marked increase in the

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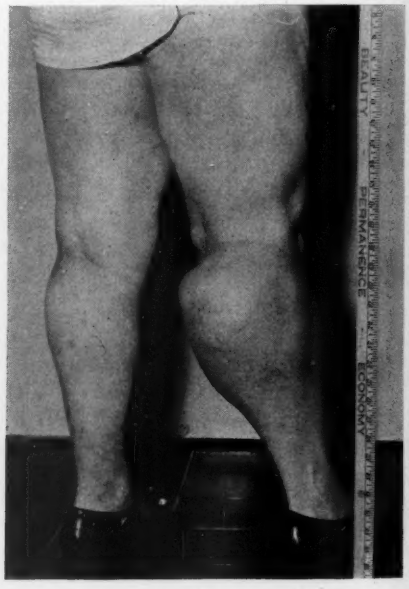

2

FIGS. 1 and 2. Standing projections of osteochondrosarcoma of right tibia as it presented preoperatively.

FIG. 3. Preoperative roentgenogram of right knee and leg showing characteristics of the osteochondrosarcoma.

proportion of calcified material. There was atrophy of the soleus-gastrocnemius group of muscles and moderate atrophy of the peroneus muscle. There was no foot drop. The pain was described as local in the tumor mass with an occasional *dart* of pain into the mid forefoot and great toe on weight bearing. She was told that amputation was the only relief feasible and a mid thigh amputation was proposed. This was refused and the patient migrated through the services of 14 physicians with many recommendations for amputation and palliation with progressively larger doses of narcotics.

In the spring of 1953, the patient was again examined and amputation was recommended but refused. The tumor had attained gigantic proportions, measuring 30 by 40 by 45 cm. It was irregularly bossed, and firmly fixed on palpation without local tenderness, increased heat, or redness (figs. 1 and 2). The knee would flex only 10 per cent of its normal range due to mechanical block. The soleus-gastrocnemius muscle group was virtually absent. Though the patient could extend the ankle to moderate resistance, she could not support her weight on the toes of her left foot. There was a foot drop with peroneal palsy, but there were no sensory changes. She complained of continuous pain in the tumor with stabbing pain into the mid leg, foot, and great toe on motion. She required 1 to 2 grains of morphine sulphate each 24 hours for relief of pain.

Roentgenograms (fig. 3) of the tumor presented a mottled mass, mostly posterior to the tibia in its upper third without involvement of the knee joint. Projections of the tumor extended laterally on both sides of the tibia which gave a horseshoe effect. The mass contained considerable calcification and ossification interspaced with areas of varying radiolucency. There was no appreciable change in any of the other osteochondromas. The roentgenologist's diagnosis was chondrosarcoma or chondromyxosarcoma.

At the patient's insistence an attempt at local excision was made, with the understanding—preoperatively—that no amputation would be done unless demanded by unexpected severance or damage of essential blood supply. The patient's general physical condition was excellent. There was no evidence of distant metastases.

On March 28, 1953, the tumor was excised locally. A curved incision (fig. 4), measuring 20 cm. in length—starting over the lower medial aspect of the left popliteal space and extending within 8 cm. of the heel—was made. The soleus and gastrocnemius muscles showed marked pressure atrophy. The tumor presented immediately beneath the gastrocnemius and was well encapsulated so that a cleavage plane could be developed with ease about each

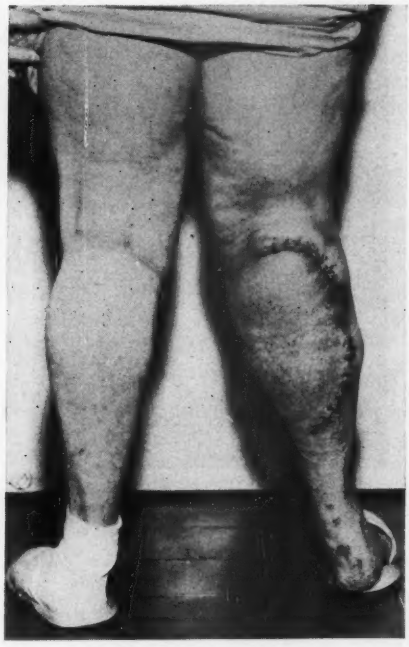

FIG. 4. Postoperative view of right leg with recent healing incision (7 days postoperatively).

quadrant of the mass. To facilitate removal the tumor was split posteriorly with scalpel and osteotome. It was attached to the cortex of the upper posterior medial aspect of the tibia over an area about 2 by 4 cm. in size. The tumor was removed with a chisel. The peroneal nerve was compressed by the tumor. A wide area of the periosteum of the posterior and lateral upper tibia was removed and the attaching cortex curetted, producing communication with the marrow cavity in an area 3 by 4 cm. in size. No extension of the tumor was found. The defect was closed by overlaying the fibrous soleus muscle to fill the dead space. Redundant skin was excised and the skin was closed with interrupted sutures of black silk. A pressure dressing and posterior splint were applied.

The patient tolerated the operative procedure without difficulty and presented no postoperative problems. Postoperative roentgenograms showed complete removal of the tumor without compromising the stability of the upper tibia (fig. 5). Weight bearing was begun in 10 days; walking with support in 14 days; and walking without support in 28 days. There was continued moderate foot drop and weakness of the ankle extensors. No sedation was required after the fifth postoperative day.

The pathologist reported that the tumor weighed 2150 Gm. Its surface was covered with bony and cartilaginous bosses; much myxoid material was interspaced with solid areas; the entire mass was encapsulated with a thick, irregular, fibrous sheath. In the central and superior areas were masses of apparent hyaline cartilage interspaced with chalky material.

Microscopically, the greater part of the tumor presented areas of degeneration, necrosis and calcification. The advancing border of tumor was always surrounded by dense fibrous tissue. The central and superior areas presented hyaline cartilage with small areas of atypical cells—some with abundant cytoplasm, others containing chiefly nuclear material—with varying degrees of staining. The distribution of lacunas in the hyaline matrix was

FIG. 5. Postoperative roentgenogram demonstrating defect in proximal posterior tibia that resulted from excision of osteochondrosarcoma. A spiked osteochondroma is present on posterior distal femur.

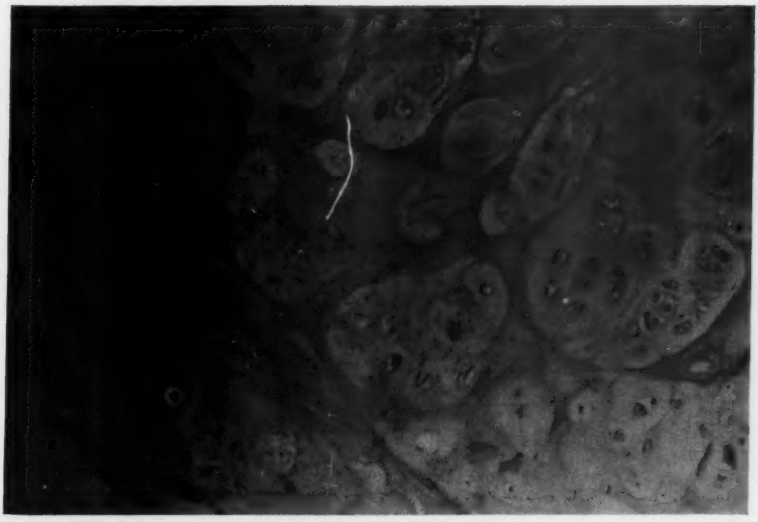

FIG. 6. Microscopic section through proliferating area of osteochondrosarcoma. To the right is noted many irregular cartilaginous islands containing multinucleated and hyperchromatic cells. To the left is normal appearing fibrocartilage.

frequently irregular, with many lacunas containing two and three cells (fig. 6). No giant cell formation or frankly cellular areas were made out in a great number of sections studied from all parts of the tumor. A separate section of tissue curetted from the base of the tumor showed the most atypical hyaline cartilage.

The pathologic report was low grade chondrosarcoma. On the basis of the pathologic diagnosis, and especially the reported active growth in the marrow area, amputation was recommended, but refused by the patient. She has returned to work and is self supporting for the first time in 11 years.

SUMMARY

In summary the characteristics of osteocartilaginous exostoses have been outlined. Their place in the formation of chondrosarcoma and the treatment of degenerative changes in osteocartilaginous exostoses have been reviewed. A case history is presented which demonstrates the usual long term, slowly progressive nature of osteocartilaginous exostoses with ultimate degeneration into chondrosarcoma.

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ANATOMY OF THE HAND

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A thorough knowledge of the anatomy and physiology of the hand is essential in order to cope intelligently with the problems that arise in its emergency treatment and definitive surgery. The relative position and level of structures in their relationship to adjacent tissues is the keynote in approaching and isolating structures without disturbing or injuring the contiguous anatomy.

The anatomy of the hand will be discussed layer by layer beginning with the skin and ending at the level of the metacarpal bones. The deep fascia lies at various levels and will be discussed accordingly.

The following outline lists structures in the anatomical layers of the hand:

Volar

1. Skin
2. Superficial fascia
3. Deep fascia (palmar aponeurosis)
4. Superficial volar arch
5. Ulnar nerve
6. Transverse carpal ligament
7. Median nerve
8. Flexor tendons and lumbricals
9. Thenar and mid-palmar spaces
10. Adductor pollicis muscle
11. Deep volar arch
12. Deep branch—ulnar nerve
13. Volar interosseous fascia
14. Interosseous muscles.

Dorsal

1. Skin
2. Superficial fascia
3. Deep fascia
4. Extensor tendons
5. Subaponeurotic space
6. Dorsal carpal arch
7. Dorsal interosseous fascia.

VOLAR SURFACE

1. *Skin.* The skin over the volar surface contains sweat glands and an abundance of lymphatics. The lymphatics and veins course to the dorsum of the hand and then up the arm. There are three types of skin markings on the hand. The coarse lines make up the flexion creases, the medium lines form cleavage lines, and the fine lines make up the papillary ridges.

A. Flexion creases (figs. 1 and 2) are formed by septa that anchor the skin to the underlying tissues, thereby creating a constant creasing of the skin at a fixed point.

(a) Finger creases are transverse lines formed by flexion at the finger joints. The distal digital crease is usually situated proximal to the distal joint. There are two middle digital creases, and the distal line usually outlines the joint. The proximal digital crease is at the level of the free edge of the web and is distal to the metacarpo-phalangeal joint.

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(b) Palmar creases consist of two transverse and three longitudinal lines. The Distal palmar crease is proximal to the metacarpal heads and runs from the ulnar border to the first web space. The Proximal palmar crease is more prominent at the radial aspect and fades at the hypothenar eminence. The thenar longitudinal line starts in common with the proximal transverse line and curves around the thenar eminence.
The middle longitudinal line starts in the first web space and runs obliquely down the palm.
The ulnar longitudinal line begins at the second web space and runs parallel to the middle line.

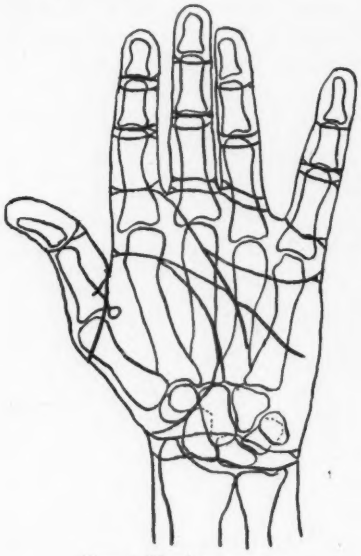

FIG. 1 Flexion creases

(Courtesy of F. W. Woods—"Principles of Anatomy of the Hand"—Williams & Wilkins Co., Baltimore, 1942)

(c) Wrist creases: The distal crease passes over the distal surface of the hamate bone. The proximal crease outlines the radio-carpal joint.

B. Cleavage or tension lines (fig. 2) are fine wrinkles dependent upon the direction of the strands of the dermal fibrous tissue. These lines run in the longitudinal axis on the volar surface and in the transverse axis on the dorsal surface of the fingers, in the transverse axis on the thenar eminence and wrist, and in the oblique axis on the dorsum of the hand.

C. Papillary ridges (fig. 2) are linear thickenings of the epidermis covering the grasping surface of the hand. The ramifications present marked individual variations.

2. *Superficial Fascia.* The subcutaneous tissue is made up of firm adherent fat lobules held in a connective tissue mesh. The fat pads protect the nerves and

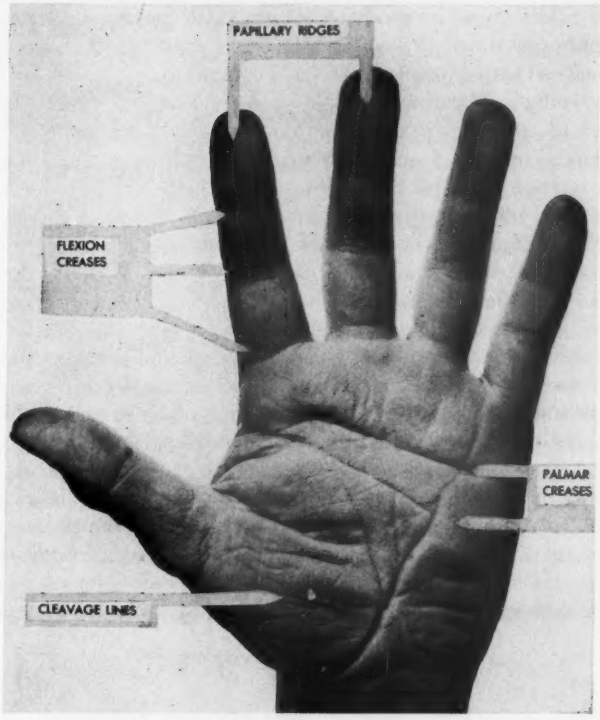

FIG. 2

vessels and appear as small round masses giving the subcutaneous tissue a coarsely granular appearance. Nerves and vessels pierce the deep fascia as single trunks and subdivide in the subcutaneous layer as they pass to the skin surface.

The palmaris brevis is a superficial muscle arising from the palmar aponeuroses and the transverse carpal ligament and inserts into the skin of the medial border of the hand, covering the ulnar artery and nerve.

The web spaces are three fatty bulges on the volar surface at the interdigital clefts. The fatty tissue bulges between the four slips of the palmar aponeuroses and are separated into dorsal and palmar compartments by the transverse metacarpal ligament. The web spaces extend proximally into the connective tissue spaces around the lumbrical muscles.

The distal pulp of the fingers. The pulp is composed of subcutaneous fat and comprises a closed pocket which is subdivided by fibrous septa into about 16 compartments. This closed space does not communicate with the tendon sheath.

3. *Deep Fascia* (A). The deep fascia is a continuation of the deep fascia of the forearm, and forms a dense fibrous ensheathing layer for the support of the structures of the hand.

The deep fascia gives rise to the following structures:

1. Palmar aponeurosis
2. Transverse carpal ligament
3. Fibrous digital sheaths
4. Vaginal ligaments
5. Palmar ligaments
6. Transverse metacarpal ligaments
7. Superficial transverse palmar ligaments
8. Cutaneous ligaments of the phalanges
9. Internal and external intramuscular septa
10. Interosseous fasciae (volar and dorsal)
11. Dorsal carpal ligament

The palmar aponeurosis and the transverse carpal and dorsal carpal ligaments will be discussed at their appropriate level.

Fibrous digital sheaths (fig. 3) are formed by strands of fascia which encircle the fingers and extend from the transverse metacarpal ligament to the base of the terminal phalanx. The sheath is attached to the borders of the phalanges and interphalangeal joints and merges proximally with the palmar aponeurosis.

Vaginal ligaments[4] are firm convex slings attached to the phalanges to form the digital tunnels (fig. 3). The fibrous digital sheath is lax as it crosses the joints of the fingers and is re-enforced by these transverse fibers in front of the proximal and middle phalanges to form two fibro-osseous digital tunnels.

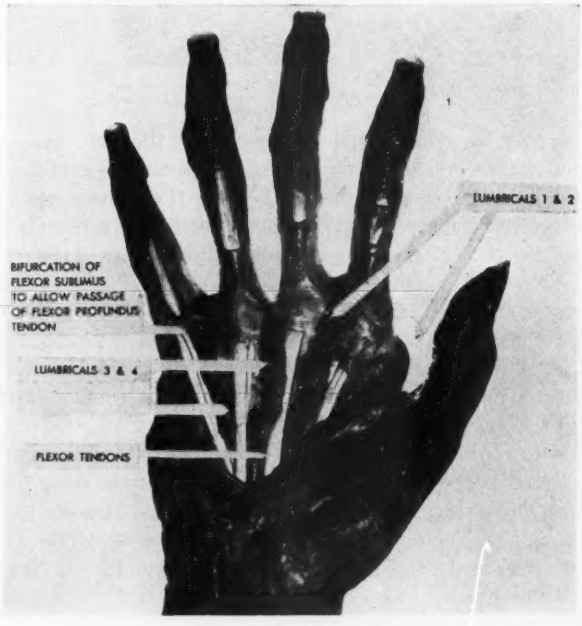
LUMBRICALS 1 & 2
BIFURCATION OF FLEXOR SUBLIMUS TO ALLOW PASSAGE OF FLEXOR PROFUNDUS TENDON
LUMBRICALS 3 & 4
FLEXOR TENDONS

FIG. 3

Superficial transverse palmar ligaments run within the skin fold of the web and are continuous with the fibrous digital sheaths.

Palmar ligaments[5] are continuous with the palmar aponeurosis and lie between the head of the metacarpal bone and the flexor tendons.

Transverse metacarpal ligaments link the palmar ligaments and metacarpal heads from the first to the fifth fingers and pass between the lumbrical and interossei tendons.

The cutaneous ligaments of the phalanges[6] are fibrous septa that course from the skin to the phalanges and interphalangeal joints for the support of the digital nerves and vessels.

The internal and external intramuscular septa[6] pass to the ventral surface of the third and fifth metacarpals, separating the thenar and hypothenar muscles from the central compartment. The posterior walls of these compartments are lined by the volar interosseous fascia.

Interosseous fascia lines the volar and dorsal surfaces of the interossei muscles.

Palmar Aponeurosis (B). The palmar aponeurosis (fig. 4) is a fibrous triangular sheet composed of longitudinal, transverse, and oblique fibers which support and protect the deeper structures of the palm.

Longitudinal fibers: The superficial fibers are continuous with the palmaris longus. The aponeurosis divides distally into four slips which cover the flexor tendons. These slips separate into two terminal branches which join the fibrous digital sheaths at the metacarpophalangeal joints and proximal phalanges. The

FIG. 4

marginal longitudinal fibers merge with the deep fascia covering the thenar and hypothenar eminences.

Transverse fibers: The deep fibers merge proximally with the transverse carpal ligament and distally form the roof of the lumbrical canals by lacing together the longitudinal fibers to form arcades between the fingers.

Oblique fibers:[2] These fibers extend to the thumb.

Superficial fibers: Fibrous septa course between the palmar aponeurosis and the skin, thereby creating adherence to the skin at the flexion creases. Vessels and nerves pierce the aponeurosis to supply the skin.

Posterior fibers: Firm septa run from the undersurface of the aponeurosis to attach to the interosseous and adductor fasciae. Distally, these septa blend with the tendon sheaths and form the walls of the lumbrical canals, thereby separating the tendons from the lumbrical muscles.

4. *Superficial Volar Arch. The superficial volar arch* (fig. 5) is covered by the palmar aponeurosis and lies superficial to the peripheral nerves and flexor tendons. It is outlined at the level of the outstretched thumb. The arch is the continuation of the ulnar artery in the hand anastomosing with the superficial volar branch of the radial artery or some other branch of the radial artery to the thumb or index finger (fig. 6).

The ulnar artery passes to the radial aspect of the pisiform bone beneath the volar carpal ligament. The superficial volar branch arises proximal to the wrist, crosses the base of the thumb, pierces the superficial thenar muscles and unites with the terminal ulnar artery.

Four common digital arteries arise from the convexity of the arch to supply the medial three and a half digits. These branches divide at the interdigital clefts into proper digital branches. Each digital vessel is joined by a volar metacarpal artery from the deep volar arch. The digital arteries run along the margins of the fingers supported by the skin ligaments, and terminate in a complex capillary network in the nail bed and pulp of the terminal phalanges.

5. *Ulnar Nerve. The ulnar nerve* (fig. 6) lies medial to the ulnar artery as it passes over the transverse carpal ligament and under the volar carpal ligament and palmaris brevis. The nerve enters the hand by passing between the pisiform and the hamate bones to divide into superficial and deep branches. The superficial branch supplies cutaneous branches to the medial one and a half fingers, a motor branch to the palmaris brevis, and a communiacting branch to the median nerve.

6. *Transverse Carpal Ligament. The transverse carpal ligament* (fig. 6) is a tough fibrous band that forms a concave membraneous arch to encase the flexor tendons, sheaths, and the median nerve. These structures traverse the fibro-osseous carpal tunnel formed by the ligament and the carpal bones. The ligament is crossed anteriorly by the palmar branches of the median and ulnar nerves, the palmaris longus tendon, the ulnar artery and the ulnar nerve.

The proximal and distal borders of the transverse carpal ligament are continuous with the deep fascia of the forearm and palm. The distal border gives origin to some fibers of the short muscles of the hand. The ligament is attached

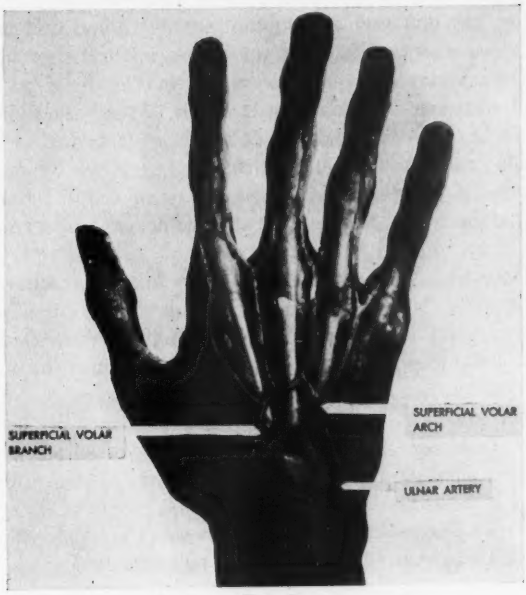
SUPERFICIAL VOLAR BRANCH
SUPERFICIAL VOLAR ARCH
ULNAR ARTERY

FIG. 5

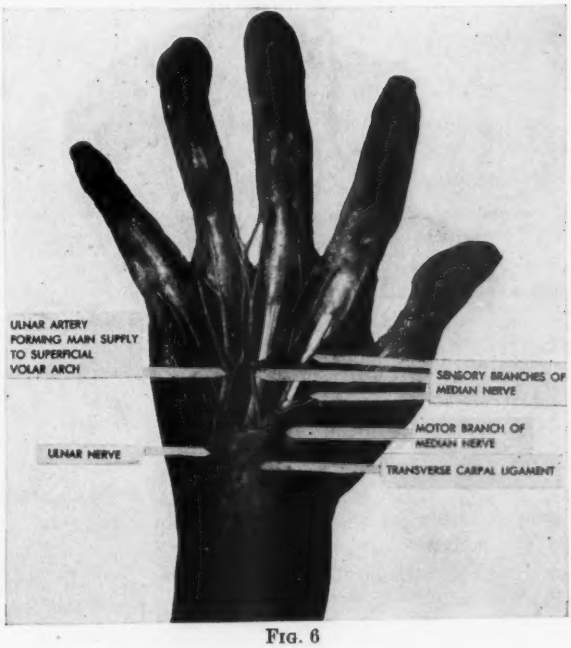
ULNAR ARTERY FORMING MAIN SUPPLY TO SUPERFICIAL VOLAR ARCH
SENSORY BRANCHES OF MEDIAN NERVE
MOTOR BRANCH OF MEDIAN NERVE
ULNAR NERVE
TRANSVERSE CARPAL LIGAMENT

FIG. 6

laterally to the navicular and greater multangular bones and medially to the pisiform and hamate bones. The proximal part is lax and stretches between the tubercle of the navicular and the pisiform bones. The distal part stretches between the ridge of the multangular and the hook of the hamate bones. A tunnel is formed for the flexor carpi radialis tendon by fibers attached to the ridge of the lateral lip of the greater multangular bone.

The volar carpal ligament is a lax band of fibrous tissue extending from the transverse carpal ligament to the pisiform bone and passes over the ulnar artery and nerve.

The thenar muscles (fig. 7) arise from the transverse carpal ligament, the tubercle of the navicular, and ridge of the greater multangular bones. They insert into the radial side of the proximal phalanx (abductor); into the entire lateral surface of the first metacarpal (opponens), and into the adjacent base of the proximal phalanx (flexor).

The hypothenar muscles (fig. 7) arise from the transverse carpal ligament, pisiform, and hamate bones. They insert into the medial side of the base of the proximal phalanx (abductor); into the medial margin of the fifth metacarpal (opponens) and into the mesial side of the proximal phalanx (flexor).

7. *Median Nerve. The median nerve* (fig. 6) enters the wrist lying medial to the flexor carpi radialis tendon and deep to the palmaris longus tendon; traverses

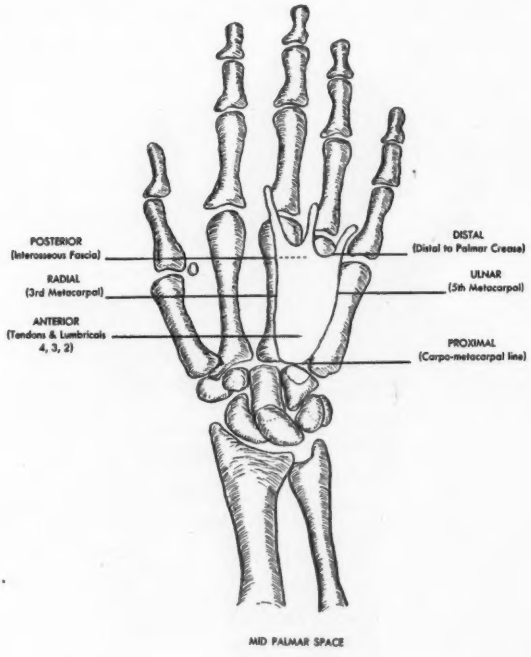

FIG. 7

the carpal tunnel beneath the carpal ligament, and emerges at the distal end of the ligament opposite the medial edge of the thenar eminence, dividing into a motor and five sensory branches. The motor branch curves around the distal border of the carpal ligament and supplies the three thenar muscles. The superficial palmar artery lies along the medial side of the nerve. Three sensory branches supply each side of the thumb and the lateral aspect of the index finger. The two remaining common sensory branches become superficial at the interdigital clefts and divide into digital branches to supply the adjacent sides of the index and middle, and middle and ring fingers. The first two lumbrical branches arise, respectively, from the lateral side of the index finger and from the contiguous sides of the index and middle fingers. Dorsal branches supply the terminal phalanx of the thumb and the distal two phalanges of the first two and a half fingers. Fine branches supply the metacarpo-phalangeal and interphalangeal joints of the thumb, index, and middle fingers.

8. *Tendons. The flexor tendons* (fig. 3) traverse the carpal tunnel; course through the palm; enter the fibro-osseous digital tunnels, and terminate at their respective points of insertion.

Carpal tunnel: The flexor sublimis tendons enter the carpal tunnel with the third and fourth tendons, lying anterior to the second and fifth tendons. The flexor profundus and flexor pollicis longus tendons lie posteriorly.

Palm: The sublimis and profundus tendons course beneath the digital slips of the palmar aponeurosis and then enter the digital tunnels at the level of the metacarpal heads.

Fingers: The sublimis tendon splits in front of the proximal phalanx into two slips to insert on the proximal margins of the middle phalanx. The profundus tendon passes through the split and inserts into the base of the terminal phalanx.

The *lumbrical muscles* (fig. 3) arise from the profundus tendons in the palm; form tendons that pass through the web spaces anterior to the deep transverse ligaments and insert into the radial aspect of the dorsal expansions just distal to the interossei insertions.

The *synovial sheaths* are lubricating mucous sheaths encasing the flexor tendons during their passage through the carpal and digital tunnels. The digital sheaths of the index, middle, and ring fingers extend to the level of the distal transverse crease. The carpal sheaths of the sublimis and profundus tendons become one common flexor sheath which extends above and below the transverse carpal ligament. The carpal and digital sheaths of the thumb and little fingers are usually continuous. The vinculae carry blood vessels to the tendons.

The *ulnar bursa* is a continuation of the sheath of the flexor tendon of the little finger that forms a common synovial membrane with the flexor tendons invaginated into this synovial cavity.

The *radial bursa* (fig. 7) is a continuation of the sheath of the flexor pollicis longus muscle that extends from the base of the terminal phalanx to a point about one inch above the transverse carpal ligament. This bursa may communicate with the common flexor sheath.

9. *Spaces.* The closed fascial spaces are potential spaces interposed between

the flexor tendons and the interosseous and adductor pollicis muscles. The two spaces are divided by a strong septum which is weakest at its proximal border, occasionally permitting communication.

The mid palmar space (fig. 7) extends from the middle metacarpal bone to the radial aspect of the fourth metacarpal bone. *Anteriorly*, it is covered by a thin fibrous sheet separating it from the tendons and lumbrical muscles of the small, ring, and middle fingers. *Posteriorly*, it is covered by a thick fibrous sheet separating it from the deep palmar arch and interossei muscles. *Ulnar side*, it is limited by a dense fibrous sheet which separates it from the hypothenar eminence. *Radial side*, it is limited by a strong fibrous septum which is attached to the median ridge on the middle metacarpal bone. *Distally*, it extends distal to the level of the distal palmar crease and sends prolongations along the fourth, third, and second lumbrical canals. *Proximally*, it extends to the level of the carpo-metacarpal joint line and sends a fine prolongation to reach the anterior surface of the pronator quadratus muscle.

The thenar space (fig. 8) extends from the middle metacarpal bone to the radial aspect of the hand. *Anteriorly*, it is covered by the muscles of the thenar eminence and flexor tendons of the index finger and thumb. *Posteriorly*, it is covered by a thin fibrous sheet which separates it from the adductor pollicis muscle and a portion of the first dorsal interosseous muscle. *Ulnar*, A strong

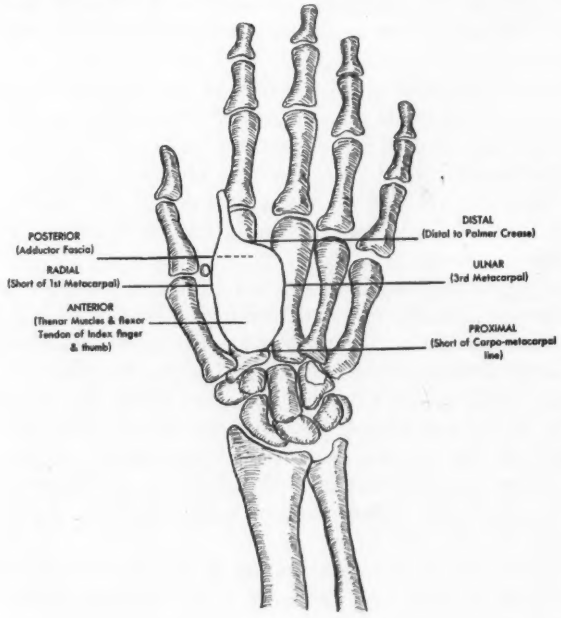

FIG. 8

fibrous sheet separates it from the mid palmar space. *Radially*, it stops short of the first metacarpal bone and lies to the medial side of the flexor pollicis tendon. *Distally*, it extends distal to the level of the proximal palmar crease, and sends a fine prolongation along the index lumbrical canal. *Proximally*, it extends to the level of the carpo-metacarpal joint line.

10. *Adductor Pollicis Muscle. The adductor pollicis muscle* (fig. 7) is a triangular shaped muscle lying behind the thenar space and anterior to the interosseous muscles. The proximal oblique head arises from three carpal and three metacarpal surfaces; the distal transverse head arises from the median ridge of the distal two-thirds of the third metacarpal bone. The tendon inserts into the ulnar sesamoid and the base of the proximal phalanx of the thumb. A slip of muscle separates from the proximal border of the oblique head and inserts into the radial aspect of the base of the proximal phalanx, together with the flexor pollicis brevis (first volar interosseous muscle).

11. *Deep Volar Arch.* The third part of the radial artery passes between both heads of the first dorsal interosseous muscle and enters the palm between both heads of the adductor pollicis to continue as the deep arch through the union with the deep branch of the ulnar artery.

The deep volar arch (fig. 10) lies on the proximal borders of the metacarpal bones and interosseous muscles. It extends distal to the transverse carpal ligament and proximal to the superficial volar arch. The arch supplies palmar digital

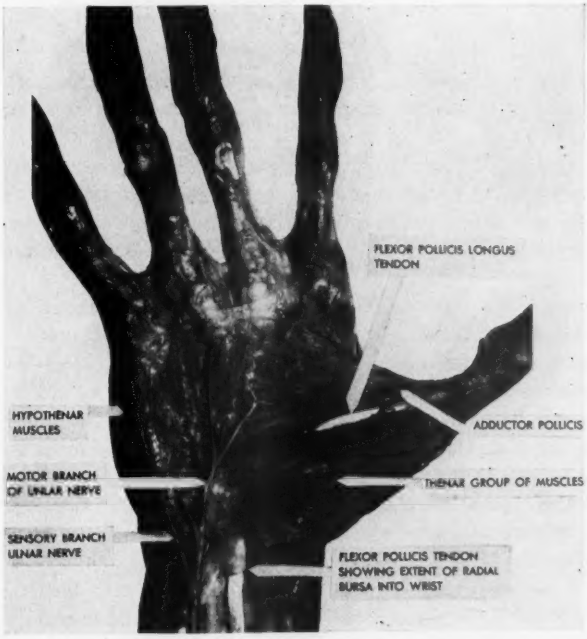
FLEXOR POLLICIS LONGUS TENDON
HYPOTHENAR MUSCLES
ADDUCTOR POLLICIS
MOTOR BRANCH OF ULNAR NERVE
THENAR GROUP OF MUSCLES
SENSORY BRANCH ULNAR NERVE
FLEXOR POLLICIS TENDON SHOWING EXTENT OF RADIAL BURSA INTO WRIST

FIG. 9

FIG. 10

branches to one and one-half digits, three palmar metacarpal arteries, three perforating arteries to join the dorsal metacarpal arteries, and several branches to the anterior carpal arch.

12. *Deep Branch—Ulnar Nerve. The deep branch of the ulnar nerve* (Fig. 7) crosses the hook of the hamate bone and crosses the palm obliquely in company with the deep palmar arch and supplies three hypothenar muscles, seven interossei, two lumbricals, the short head of the flexor pollicis brevis, adductor pollicis, and several joints.

13. *Volar Interosseous Fascia. The volar interosseous fascia* lines the interosseous muscles and becomes thickened at the metacarpal heads, thereby forming the transverse metacarpal ligament.

14. *Interosseous Muscles. The volar interossei* are three short muscles that arise by single heads from the metacarpal bones and adduct the index, ring, and little fingers. The deep head of the flexor brevis can be considered the first volar interosseous muscle as it arises from the ulnar side of the base of the first metacarpal bone and inserts into the ulnar sesamoid, along with the adductor pollicis. The second interosseous muscle inserts on the ulnar aspect; the third and fourth insert on the radial aspects.

The dorsal interossei (figs. 11 and 12) are four short muscles that arise from the adjacent sides of the metacarpal bones and abduct the fingers. The first and second insert on the radial aspect, the third and fourth insert on the ulnar aspects.

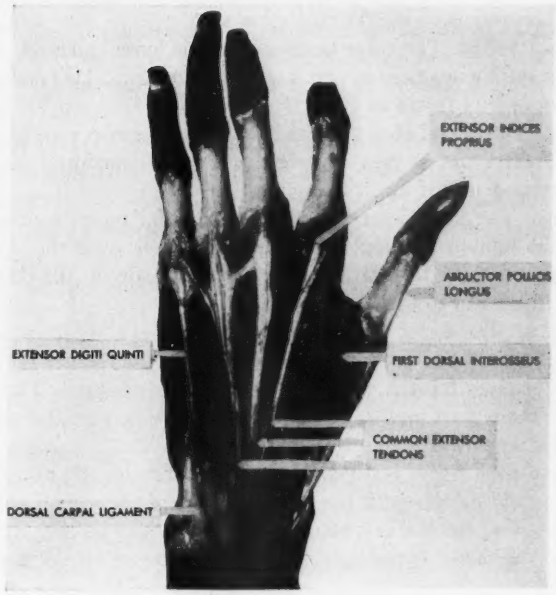

FIG. 11

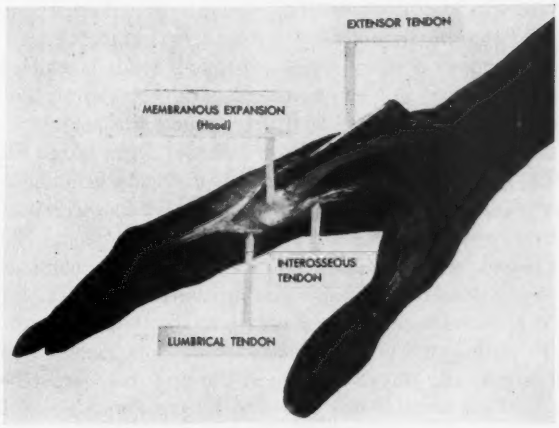

FIG. 12

The interossei tendons form at the level of the metacarpo-phalangeal joint; pass beneath the deep transverse ligament, and insert into the lateral tubercle of the proximal phalanx and expand on the dorsum into the lateral slips of the extensor aponeuroses.

DORSAL SURFACE

2. *Superficial Fascia.* The subcutaneous tissue is loose and thin, and contains adipose tissue, cutaneous nerves, and superficial vessels. This layer merges with the palmar superficial fascia at the web spaces.

Ulnar nerve: The dorsal sensory branch of the ulnar nerve arises at the lower third of the forearm and courses over the distal end of the dorsal carpal ligament to supply one and a half fingers.

Radial nerve: The superficial branch of the radial nerve courses under the brachio-radialis tendon to supply the skin and fascia over the dorsum of the wrist and dorsum of the thumb. Five digital nerves supply the thumb and two and one-half fingers up to the proximal joints.

3. *Deep Fascia. The dorsal carpal ligament* (fig. 11) is a fibrous condensation of the deep fascia by oblique fibers which send septa to the radius and ulna forming fibro-osseous tunnels for the tendons and synovial sheaths. The ligament is situated at a more proximal level than the transverse carpal ligament and is crossed by the superficial veins and sensory nerves.

The intertendinous fascia is a continuous bilaminar layer[1] connecting the extensor tendons and merges with the dorsal surface of the second and fifth metacarpals. Proximally, the fascia is continuous with the dorsal carpal ligament and periosteum of the carpal bones, and distally with the capsule of the metacarpophalangeal joints.

4. *Extensor Tendons. The extensor tendons* (fig. 11) of the hand are arranged into a superficial and a deep layer. The superficial tendons are the extensor digitorum communis and extensor digiti quinti. The deep extensors are the three extensors of the thumb and the extensor indicis proprius.

The extensor communis muscle separates into four tendons which pass beneath the dorsal carpal ligament in a compartment with the extensor indicis proprius. The common tendons of the index and little fingers are joined on their medial side by the extensor indicis proprius, and extensor digiti quinti. Three oblique bands unite the medial tendons proximal to the metacarpophalangeal joints.

The extensor digiti quinti tendon passes in a groove in a compartment of the dorsal carpal ligament and splits into two parts on the dorsum of the hand. The lateral slip is joined by the fourth tendon of the extensor communis and both are inserted on the dorsum of the proximal phalanx.

The abductor pollicis longus tendon emerges in the lower forearm between the radial extensors of the wrist and the common extensors; passes under the carpal ligament and inserts into the radial side of the first metacarpal bone.

The extensor pollicis brevis tendon passes under the dorsal carpal ligament and over the radial artery; courses over the dorsal surface of the first metacarpal and inserts into the base of the proximal phalanx.

The extensor pollicis longus tendon occupies a special compartment as it passes under the dorsal carpal ligament. It extends obliquely across the hand; crosses the radial extensors and radial artery, and inserts into the dorsum of the second phalanx.

The extensor indicis proprius tendon passes under the carpal ligament in a compartment with the communis tendons and lies on the mesial side of the common tendon and inserts into the membraneous expansion of that tendon on the dorsum of the proximal phalanx.

The extensor tendons are flattened and form a hood over the metacarpal head which is closely adherent to the capsule. The expansion separates into a middle slip which passes to the base of the middle phalanx, and two lateral slips which are joined by the interosseous and lumbrical tendons and extends to the base of the distal phalanx.

As the extensor tendon extends the metacarpo-phalangeal joint, the hood moves proximally and the lateral slips of the interossei and lumbricals extend the interphalangeal joints.[3] As the extensor tendon relaxes, the hood moves distally permitting the interossei and lumbricals to flex the metacarpo-phalangeal joints by means of slings of transverse fibers that arch over the proximal phalanges.

5. *Dorsal Subaponeurotic Space. The dorsal subaponeurotic space* is a triangular subfascial space between the extensor tendons and the dorsal interosseous fascia. This space contains loose areolar tissue and is narrowest at the wrist and widest at the metacarpo-phalangeal joints.

6. *Dorsal Carpal Arch. The dorsal carpal arch* is formed by dorsal branches of the radial and ulnar arteries, between the proximal and distal row of the carpal bones. The arch sends three dorsal metacarpal arteries which divide into small dorsal digital branches. The dorsal metacarpal arteries are joined by perforating branches to the deep palmar arch.

7. *Dorsal Interosseous Fascia. The dorsal interosseous fascia* is thin and fuses with the metacarpal bones. Distally, the fascia ensheaths the tendons of the interossei and merges with the dorsal tendinous expansion.

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EDITORIAL

PHLEBOTHROMBOSIS ASSOCIATED WITH CANCER OF BODY AND TAIL OF PANCREAS

Present efforts to emphasize the importance of early cancer diagnosis are proving fruitful with respect to those tumors recognizable in the early stages. This is particularly true of cutaneous and mammary cancers. It is also true of malignant lesions of the gastrointestinal tract. Routine chest surveys are bringing a larger number of lung tumors to the surgeon while still operable. Space consuming lesions of the brain and spinal cord are being discovered earlier, thanks to the increased use of the electroencephalogram and to improved myelography.

Unfortunately cancers of the liver and pancreas continue to defy attempts at early identification, and those in which primary origin is in the body or tail of the pancreas are almost always advanced before they become symptomatic. The latter group produces no significant physical signs and roentgenologic studies are negative unless the tumor has reached sufficient size to distort the duodenal loop or displace the dependent portion of the stomach. When such conditions prevail the tumor is almost invariably inoperable. Epigastric pain is the most consistent early symptom but is in no sense diagnostic. A more hopeful early clue is the development of peripheral phlebothrombosis. Sproul first called attention to the relationship of multiple phlebothromboses to such tumors after a study of 4258 successive necropsies in which she found 125 instances of the syndrome. These observations have since been confirmed by several independent investigators and it is now generally accepted that such tumors are responsible for some alteration in the coagulability of blood to account for the thrombosis. The exact mechanism of this change is obscure but it has been observed that these tumors are frequently mucin-producing carcinomas in which the cells produce intracellular as well as extracellular substances. It has been postulated that such products may in some way affect the blood clotting factors. Trypsin, when present in high concentration, affects coagulation by destroying thrombin. It may be that a deficiency in enzyme is responsible for the development of multiple thrombi in carcinoma of the pancreas. The latter theory fails to explain the greater incidence of thrombosis in association with carcinomas of the body and tail as compared to those located in the head of the pancreas, a contradiction which may be explained by the altered liver physiology due to the biliary obstruction which so frequently occurs with tumors of the pancreatic head.

Further knowledge of this problem will depend on studies of the blood-clotting mechanism during the life of a patient suspected of having the syndrome. Detailed pathologic and chemical studies of the neoplastic tissue obtained at autopsy from such patients might furnish information concerning the presence or absence of a specific substance responsible for the altered blood clotting. Identification of such factor or factors might well provide an early clue to the existence of these obscure pancreatic malignancies.

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BOOKS RECEIVED

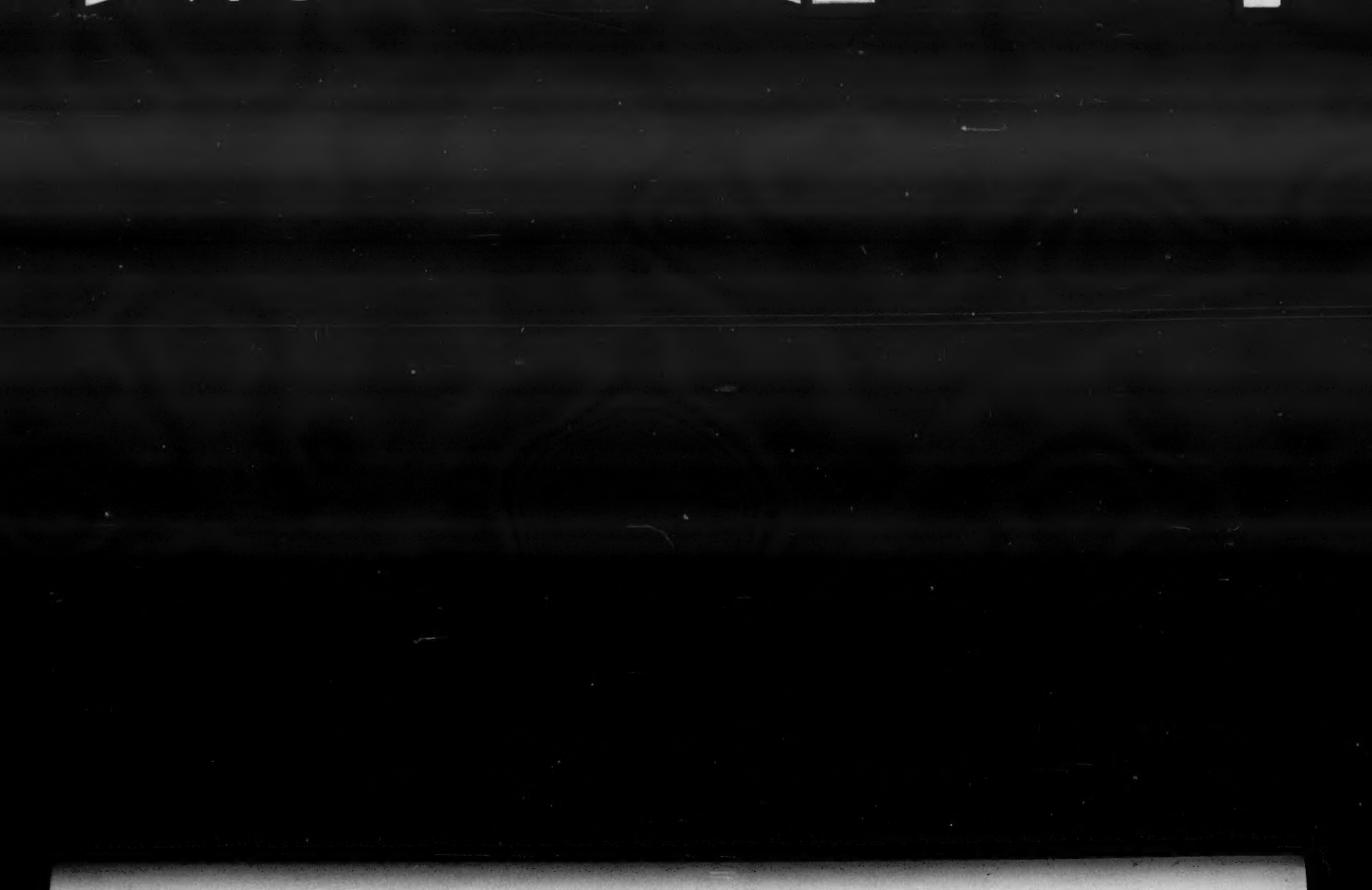
Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

The Book of Health, A Medical Encyclopedia for Everyone. Compiled and Edited by RANDOLPH LEE CLARK, JR., B.S., M.D., M.Sc. (Surgery) Houston, Texas. Director and Surgeon-in-Chief, The University of Texas, M.D., Anderson Hospital for Cancer Research; Professor of Surgery, The University of Texas, Post-Graduate School of Medicine; Fellow of the American College of Surgeons; National Consultant in Surgery, United States Air Force; and RUSSELL W. CUMLEY, B.A., M.A., Ph.D., Houston, Texas, Director of Publications, the University of Texas, M.D., Anderson Hospital for Cancer Research; Professor of Medical Journalism, The University of Texas Post-Graduate School of Medicine, Houston-New York, Elsevier Press, Inc. $10.00.

Handbook of Differential Diagnosis. By HAROLD THOMAS HYNEAN, M.D., Author of "An Intergrated Practice of Medicine." Philadelphia, London, Montreal, J. B. Lippincotto Company, $6.75.

A Guide to Medical and Dental Photography. By KENNETH S. TYDINGS, s.p.e., New York, Greenberg, Publisher, $1.95.

ERRATUM

The printer regrets the error which occurred in the November Journal, Volume 19, Number 11.

Figures 4 and 5 on pages 1041 and 1042 were printed in reverse positions in the article "Carcinoma of the Rectum" by Garnet W. Ault, M.D.